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THE OPHTHALMOLOGICAL OBSERVATIONS OF HUGHLINGS JACKSON AND THEIR BEARING ON NERVOUS AND OTHER DISEASES.[1]

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Gentlemen,—Let me first in one word thank you for the great honour you have done me in electing me to what I consider the highest position to which one who has worked at neurology may aspire. It is not easy for anyone to wear with grace and distinction the "giant's robe" which the President of this Section must necessarily assume—for my predecessors have all been eminent, and not a few of them great; but such a robe may serve to hide defects and deformities, and at least I shall hope to do nothing unworthy of it, and to hand it over intact and untarnished to a more worthy wearer.

I do not think it can be easy for anyone who was not brought intimately in contact with him to understand the feelings of reverent affection which all of the younger generation who had enjoyed that good fortune always entertained for the great personality of Hughlings Jackson. It may seem presumptuous in me to choose such a subject for my address as that which has been announced, but I should urge that I am not about to criticize, and what I have to say will be more in the nature of an attempt to appreciate some of his work. I trust that what I shall say may have the effect of stimulating interest in his writings so that their wonderful wealth of clinical observation, their accurate scientific method, and their broad philosophical grasp may be better and more generally recognized than they have been as yet.

Jackson's writings are best considered in groups. One of his earliest and most lasting interests was undoubtedly in connexion with ophthalmological and ocular conditions. These ophthalmological

[1] Presidential address delivered at the opening meeting of the Neurological Section of the Royal Society of Medicine, October 28, 1915.

interests were, of course, intimately bound up with his interest in the symptomatology of intracranial tumours, and most of his early writing has reference to these subjects. Speech and the alteration of that faculty by disease naturally appealed to his philosophical mind, and I feel sure that all neurologists feel indebted to Dr. Henry Head for the work he has done in placing Jackson's position in reference to this important subject clearly before neurologists. Another division of Jackson's work had reference to paralysis, hemiplegia, chorea and the syphilitic diseases of the nervous system. Still another—intimately associated with the last—dealt with epilepsy in its varied phases, another with vertigo and associated ear conditions. His work on the evolution and dissolution of the nervous system was really a series of philosophical treatises, and his suggestions with reference to the study of disease of the nervous system were severely scientific and yet intensely practical. Among the most interesting of his papers are those designated "Neurological Fragments," and they are, as their name implies, fragments of what one might call intensive neurological investigation and teaching, dealing with an exceedingly varied class of subjects. I need hardly say that this division of subjects is not Jackson's. It is one for which I am entirely responsible, and, as you can understand, one division may overlap another, and many papers find a natural place in more than one of the divisions. But it is convenient to divide his writings in this way so long as we regard the divisions merely as a convenient series of pigeon-holes, and to-night I propose to consider that division which has reference to ophthalmological subjects. This, as will be evident from what I have said, must naturally lead me to refer occasionally to writings which more particularly deal with the symptomatology of intracranial tumours.

The invention of the ophthalmoscope marks an epoch in medicine. It is nearly always associated with the great name of Helmholtz, but it should be remembered that four years before Helmholtz described his instrument, the ophthalmoscope had really been invented by Babbage, and was only laid aside because an ophthalmic surgeon to whom he showed it failed to discern its possible usefulness, and gave him no encouragement. It is interesting to know that the modification of Helmholtz's instrument soon approximated to the form originally invented by Babbage, and it is this form of instrument, with, of course, modifications and improvements, which is now in general use. Dr. Jackson's friendship and early association with Mr. Jonathan Hutchinson—they were both Yorkshire men, both apprenticed in York,

and both held resident posts, not simultaneously, in that city—was no doubt the cause of his becoming early acquainted with Moorfields Eye Hospital, where he worked as Mr. Hutchinson's clinical assistant for several years, and his work here gave an extraordinary stimulus to his interest in ocular and ophthalmological conditions. He has put it on record that he considered it the luckiest thing in his medical life that he began the scientific study of his profession at an ophthalmic hospital [2]. Much of his earliest writings—the result of observations while at Moorfields—are published in the "Moorfields' Hospital Reports," and are mostly concerned with ophthalmoscopic observations. But these observations had almost always reference to the association of ophthalmoscopic changes with medical disease—especially disease of the nervous system—and he was never tired of insisting upon the necessity of routine ophthalmoscopic examination in all such disease.

In an early paper he [21] says: "My object in writing this article is simply that I may contribute from my own field of clinical work a few observations on a class of symptoms, the meaning of which it is most desirable to make out for the sake of advancing our knowledge of diseases of the nervous system. This renders it necessary that I should address myself both to the ophthalmologist and to the general physician, and thus I run a double risk of criticism. If in the slightest degree I do something towards bridging over the special and perhaps somewhat isolated department of ophthalmic medicine with the more general one, I shall have done all I wish to do." The paper to which this is a preface is devoted to a study of defects of sight in brain disease. In this attention is directed to several interesting points. The occurrence of the two kinds of atrophy, one in which the nerve gradually whitens, the other in which it becomes white after certain acute changes, is mentioned, causing what was then called "amaurosis." The difficulty is mentioned of following up and carefully observing such cases because of their tendency to go from one hospital to another, according to the nature of the symptoms. The sudden failure of sight which occurs in some cases of cerebral disease is referred to. Incidentally, he alludes to a danger with which most of us are probably familiar, viz., that an ophthalmological examination, or even treatment, is sometimes blamed for loss of sight. Thus one patient ascribed his loss of sight to a blister which had been applied; another to the use of galvanism; and a third became blind soon after an ophthalmoscopic examination, and ascribed the loss of vision to the examination. In the same paper attention is directed to the headache and vomiting which occur with

intracranial tumour, and the danger of mistaking such symptoms for those of gastric disease. Further, the importance and usefulness of ophthalmoscopic examination in cases of hemiplegia, especially for evidence of Bright's disease, and in cases brought in comatose, are insisted upon.

The interest of these early papers of Dr. Jackson's is of two kinds. They embody records of observations of phenomena which were then novel, and the significance of these is set out with a clearness and completeness which left little for subsequent observers to do. But they also contain reflections and remarks which make one feel that the work as a whole is pervaded by a philosophical spirit which breathes life into the dead bones of mere clinical observations. Thus at the beginning of one of the sections of this paper he apologizes for speaking too much of some symptoms which are out of the scope of a journal of ophthalmology [23]: "Much of the interest, however," he says, "depends upon the number of symptoms which can be put together in an intelligent order. Amaurosis may be studied at once with too much intensity and too little breadth. It is to the ophthalmologist a disease of so great importance, calling for particular action on his part, that he may underrate its significance as a symptom in general conditions of the system. But it is when it occurs with other phenomena that we are most likely to discover what optic neuritis means." He then goes on to say that his object in writing is to urge physicians and ophthalmic surgeons to do more work together. Thus if amaurosis and defect of speech are found in the same patient, both the symptoms must be studied by one person. The ophthalmic surgeon can scarcely confine his thoughts to a single sense apparatus, however much his practice may be limited to the defects of that one. To the physician defect of smell, although it may be less important, is of equal significance with defect of sight. Hemiplegia with its allies is the extra-ophthalmological symptom which chiefly deserves the consideration of ophthalmologists because of the frequent ocular or visual anomalies associated with it.

"Odd as it may at first sight seem," he goes on to say, "I feel more and more convinced that there are important reasons why ophthalmologists and those physicians practising in insanity should do much work in common. Not because defects of sight are particularly associated with mental symptoms, nor because insane people may be liable to defects of sight, but because the special study of the evolution of movement (and sensation) is, I think, best begun at ophthalmic

hospitals. And so far as we can know anything definite of mind, it is, I suppose, made up of sensory and motor phenomena, the functions of a series of anatomical possibilities in the cerebrum in correspondence with its wide environment, as well as in the spinal cord with its narrow environment. . . . Now nowhere could we better begin to study intelligent movements of muscles than at ophthalmic hospitals. . . . The study of the higher movements, as for words, will, I feel sure, derive great help from the laws of muscular motion which ophthalmologists have been or will be able to establish in their own department of medical practice."

He then outlines a method of medical work: "Were I free," he says, "to work in this metropolis at the physiology of movement, from gross muscular movements towards thought, in the way I should prefer —supposing, to begin with, an ordinary general medical and scientific knowledge—I should study first in the wards and in the out-patient room of a general hospital such symptoms as hemiplegia, chorea, and epilepsy; next, at an ophthalmic hospital, paralysis of the cranial nerves and defects of sight; next at a special hospital for such symptoms as those of locomotor ataxy and certain epileptiform seizures; then at an orthopædic hospital for infantile paralysis and for so-called contractures. Now I would study specially deviations from healthy movements of the eyeballs, and the rudimentary delusional effects they cause. I would, after this work, return to the general hospital, to study defects of speech, delirium, apoplexy, coma, &c. Varieties of defects of speech I should compare and contrast, from difficulty of articulation to incoherence. At this juncture a careful study of laryngeal and throat disease would be most desirable.

"Of course, if anyone were to work at different sections of medical practice, so as merely to add isolated series of facts to one another, he would really make little progress in cultivating his own mind. Such a man would dwell with exaggeration—hurtful to his own organization of medical knowledge—on amaurosis as a defect of sight, and too little on it as a defect of a highly specialized part of universal sensation. He would not improbably neglect altogether such symptoms as defects of smell, which defects, however, are really quite as significant incidents in the revelations which disease makes of function as defects of sight are. Such a student would be altogether unprepared for the last and the very highest medical study, viz., of insanity. Special work at diseases of the mind should, I feel convinced, be begun only after a large real experience of all the special phenomena of motion and

sensation that damage to any parts of the nervous system does or may give rise to. I say a real experience, as I suppose a collection of numberless facts, however accurately gathered, is not held to be of itself a real experience. Unless a man can put the particular phenomena he himself sees under more general laws, or unless he tries to do this, he can scarcely be said to know or to be studying a thing in any very valuable sense. The knowledge the ophthalmologist has of muscular disorders of the eye, the knowledge the physician has of defects of articulation, of chorea, and of epilepsy, and that the psychologist has of incoherence and delusions, should aim to be physiological units, each different, but each related to a wide common knowledge of such laws—present or in progress—as those of the evolution of sensation and movement in organisms."

In 1863 Wordsworth [5] had drawn attention to one particular form of amaurosis occurring in tobacco smokers, and Jackson [14] took the opportunity in considering this of raising the question of the rôle of sex in disease, especially in disease of the nervous system, and of offering suggestions for its investigation. Wordsworth had noted the much greater frequency of optic atrophy in men than in women. Hutchinson from a large mass of material had made the same observation. This, as Jackson says, while pointing to some greater liability in the male to optic atrophy, it would be unreasonable to ascribe to tobacco-smoking until more positive and negative evidence had been obtained. He suggested, however, widening the inquiry as to the influence of sex in disease by considering two other diseases—progressive locomotor ataxy and general paralysis of the insane—both of which are diseases of the nervous system rarely found in women [15].

"The widening of the subject," he said, "need not confuse the inquiry by making it more complex; for on a scale sufficiently broad to avoid error, especially the error of attaching too much importance to accidental circumstances, amaurosis can only be studied by an ophthalmologist, progressive locomotor ataxy by those practising generally, and general paralysis by physicians to large asylums. Although, then, there would be more work there would be more workers; and yet when working singly the workers would have a bond in unity of purpose, as their ultimate object would be the same. For although the diseases of the nervous system to be investigated are widely different as groups of physiological symptoms, the pathological question—or at least, the most important part of it—is as to the effect of tobacco-smoking and other sexual habits, peculiarities, or vices, in

leading directly or indirectly to degeneration of the elements of a certain tissue—the nervous."

I mention this paper and its suggestions as indicating how widely awake Jackson's mind was to the bearing of recognized ophthalmological conditions on the etiology of nervous disease generally.

Jackson had written as early as 1863 [22] on the existence of optic neuritis without obvious defect of sight, and had frequently repeated this observation. Hutchinson [16] had drawn attention, in 1867, to Jackson's observations, for apparently they had been doubted or even ignored, and Jackson [17] writes that Clifford Allbutt's experience confirms his observations. He goes on to say with quite characteristic modesty: "So far as the observer is concerned the discovery is a very small matter. Any physician who will use the ophthalmoscope by routine must quickly discover the fact. Yet the obvious deduction is really a very important one in practice. This is embodied in the following quotation—italicized in the original—from a paper which I published in 1863 [22]. '*It is, I submit, imperative, in all cases of severe cerebral disease, at all events in cases of an acute kind, to examine the eyes with the ophthalmoscope, whether the patient complains of defect of sight or not.*"

"I should now make the statement much stronger by adding, '*even if he affirms that he can see well, and if he read small type readily.*'

"I have occasionally had the somewhat painful feeling that the accuracy of my ophthalmoscopic examination has been doubted by physicians whose opinion I highly value, when I have declared that patients with severe cerebral disease, who seemed to see quite well, had inflamed optic disks." This point Jackson had to insist upon many times before it was accepted, and we are apt to forget that what to us is now commonplace required such reiteration and insistence.

A question which was frequently discussed at this time was, Does disease of the cerebellum lead to loss of sight? Brown-Séquard had stated that it was certainly often associated with this, and he expressed the opinion that it was the result of reflex action from some irritant. Jackson [1] discusses the question, and clearly expresses the opinion that disease of the cerebellum, *per se*, does not produce blindness, any more than disease of the cerebrum, but that tumour in either region may and often does, by setting up optic neuritis—"the result of a local encephalitis." "Whilst destruction of no part of the cerebrum or cerebellum leads to loss, not even to defect of sight, irritation by tumour in any part of either may lead to inflammation of the optic nerves, on which inflammation, blindness usually—not always—follows."

At this stage, in 1871 [18], Jackson published a lecture on optic neuritis in intracranial disease, in which he sums up in a succinct form much of what he had been teaching for several years. He points out the value of the ophthalmoscope in diagnosis: that morbid ophthalmoscopic signs are rarely absent in amaurosis from cerebral disease, that optic neuritis is the commonest ophthalmological condition in cases of cerebral disease, and is as important in the diagnosis of cerebral disease as is hemiplegia. He then describes the stages of optic neuritis—the onset, the second stage with great swelling, the third in which atrophy is commencing, and the fourth that of permanent atrophy. He also again, what he earlier insisted upon, points out that in some cases of optic neuritis there is no defect of sight, that optic neuritis is almost invariably double, and that paroxysmal failure of sight occurs in cases of optic neuritis. Occasionally, also, it is pointed out, there is rapid and permanent failure of sight. With reference to the nature of the intracranial disease most often associated with double optic neuritis he makes two statements:—

(1) There is usually a gross change, or "coarse" disease, a lump of something, an adventitious product.

(2) Double optic neuritis does not point to any particular *kind* of coarse disease, but simply to coarse disease of *some* kind. There are certain conditions under which double optic neuritis scarcely ever occurs, e.g., chronic and general convulsive attacks, a condition of hemiplegia from local softening, &c., and he points out that optic neuritis is not a localizing sign, and then discusses the different theories as to the manner in which an adventitious product in the cerebrum leads to optic neuritis. He concludes by begging his readers to remember three things: (1) That optic neuritis frequently exists when the patient can read the smallest type: (2) that the ophthalmoscopic appearances vary extremely in degree in cases of adventitious products within the cranium, and that the appearances vary much at different stages in the same case; and (3) that one should never omit to use the ophthalmoscope when a patient has severe and continued headache.

During the next few years Jackson continued from time to time his ophthalmological contributions. His "Physician's Notes on Ophthalmology" [25] are a storehouse of interesting records of many cases carefully and systematically observed, and of even more valuable suggestions. He also published anomalous cases [26], one of large cerebral tumour *without* optic neuritis, another [27] of double optic neuritis without cerebral tumour, and he also recorded [32] a case of

recovery from double optic neuritis. It is clear, as will be seen, that he recognized the curious variability in the incidence of ophthalmological signs of severe cerebral disease; and that (1) intracranial tumour may be present without optic neuritis; (2) optic neuritis may be present and yet no tumour be found; and (3) optic neuritis does not necessarily result in blindness, although there is usually great danger of its leading to impairment of sight, and yet that occasionally it passes off without causing any impairment of vision.

The Annual Oration to the Medical Society of London [2], which he delivered in 1877, is perhaps one of the most characteristic and most finished of Jackson's addresses. It is an interesting combination of that scientific knowledge, clinical observation and philosophical suggestion to which reference has already been made. It sums up in a very clear way much of what Jackson had been engaged in teaching for many years, and more especially it dwells upon the work of the ophthalmic surgeon as a training in discipline and surgical skill, and as affording opportunity for observation of ocular palsies and their effects, of the anomalies of refraction, of the actual state of a nerve and its termination, and of the condition of the blood-vessels and the retina generally. He emphasizes the importance of recognizing the effects which refractive errors may produce. Thus astigmatism may produce "confusion of vision" because of the difference in the distinctness in different meridians; hypermetropia may cause, and has actually caused, symptoms simulating those of brain disease, such as headache and squinting, and observation of hypermetropic disks is of the utmost importance because of the close resemblance they bear to the inflamed disk associated with intracranial tumour. The phenomena of what was then called diphtherial amaurosis as they are revealed to the ophthalmic surgeon are also dealt with, and their relationship to affection of the ganglia is pointed out, and the interesting question is raised whether in diphtheria there is, as affecting hearing, something analogous to paralysis of accommodation in the eye.

The study of paralysis of the ocular muscles is considered. This naturally leads to a consideration of vertigo generally and also of defects of co-ordination. The vertigo which occurs from paralysis of an ocular muscle is not, it is pointed out, the result of diplopia, but of erroneous projection. A patient suffering from paralysis of the right external rectus, if the left eye is covered and he is asked to touch an object placed to the right of the middle line, will miss the object, his hand going to the right of it. That is, the patient judges by the activity of the centre for

movement—"the hand is *misinformed*," to speak metaphorically, or to use an incoherent simile, "makes a false step." It is not difficult to see the analogy between such an action and actual reeling. During walking there will be many actual false steps due to the misleading information which the centre for the impossible movement of the eye supplies. It is inaccurate to say that vertigo *causes* disorder of co-ordination: it is itself disorder of co-ordination. The only difference between vertigo *without* manifestation and vertigo *with* the outward manifestation of a "reel" is one of degree. In the former there are slight central changes and in the other strong central changes, leading to peripheral effects. There is thus an analogy between those two conditions, or rather degrees, of vertigo and the two conditions of speech—speaking internally and speaking articulately.

He then proceeds to draw a distinction between sensation and the physical process occurring along with it: "The use of the word sensation both for a state of consciousness and for a state of the nervous centres produces the same kind of confusion as the use of the expression that the sun rises in the east would do in an exposition of the movements of the solar system!"

"The facts of ocular vertigo, with other allied facts from other departments of medical practice, throw light on psychico-physical operations. Speaking generally, they help to show that ideas arise, not only during energizing of sensory centres, which everybody admits, but during energizing of motor centres, which scarcely anybody seems inclined to believe. We have seen that the patient judges of the positions of objects by central discharges which do not actually displace his retinal images. Now the extension of an object is really made up of relations of innumerable positions. To give a particular illustration, the facts of ocular vertigo are of inestimable value in supporting the doctrine that the anatomical substratum of a word is a motor, an articulatory process."

Another aspect of the matter is next considered—viz., that in some cases of paralysis of ocular muscles the size of the object is altered. The fact is referred to that if we impress the retina with a scarlet object the after-image will be altered in size with accommodative efforts. There is in this phenomenon, of course, a motor change only, for the retinal area affected is unaltered. The shape of objects also, it is pointed out, can be altered in some degree. Thus if we obtain the after-image of a circle and project this on to an inclined sheet of paper, the spectral circle becomes oval. The case is also mentioned of a

medical man subject to migraine, who observed that he could alter by accommodative efforts the size of the visual spectra which preceded his headache. And in some cases of epilepsy there is a warning in the alteration of the size of objects: mostly they get bigger and nearer—"the walls of the room seemed to come nearer," as one patient expressed it. All these cases, except probably that of epilepsy, it is claimed, show that estimation of the size of objects depends upon activity of a motor element only. And the fact that in paralysis of the ciliary muscle by atropine or by disease objects appear smaller shows that activity of motor centres may cause alteration in the size of an object when actual movement is impossible.

"These facts," he goes on to say, "like those given in speaking of vertigo, show plainly, I think, the inseparable connexion of motor activity with sensory activity in ideation. They show that, whilst the colour—the secondary or dynamical quality of an object—is a sensory affair, its size and shape, its primary or statical quality, is a motor affair. If so, then ophthalmological facts are of inestimable value, not only as bearing on an important psychological problem, but as bearing on the most important of all questions whatever in psychology; they demonstrate that the estimation of the extension of objects is due to motor activity; and they show that activity of motor centres will suffice; and thus they lend support to the doctrine that, in remembering the shape of an object, there is slight activity of central motor centres just as much as there is slight activity of sensory centres in remembering its colour."

Similarly in reference to locomotor ataxy he argues against the hypothesis of a "special co-ordinating centre." He explains the ataxy by supposing that the current developed does not—on account of diseased tracts—react with full effect on the muscle most concerned with accurate locomotion, the peroneus longus, but overflows into the tibialis anticus and so produces in it over-action—really the same result as what is called "secondary deviation" in certain conditions of ocular paralysis. There is, in short, over-estimation of the intended, but underdone, peroneal movement and actual overdoing of the tibialis anticus movement. An analogous condition had been pointed out by Duchenne in the case of a hand wasted in regard to its flexors. When such flexors are atrophied, and a strong attempt is made to close the fingers, the actual effect is over-extension of the fingers, the current intended for the paralysed flexors actually overflowing into the extensors.

With reference to sensory affection of the eyes, it is pointed out

that hemiopia is a sensory symptom which is the strict analogue of the motor symptom, lateral deviation of the eyes, and it is frequently associated with hemianæsthesia. Sight sensations, it is pointed out, are more frequently precursors of epileptic attacks than any other special sense warnings except touch. Smell is next, but auditory warning is rare. That sight should be the most frequent is what one would expect, for most ideation is carried on in visual ideas. A patient may have sight sensations, e.g., coloured vision, long before he has epileptic attacks ushered in by such sight sensations.

He then proceeds to refer to the use of the ophthalmoscope, pointing out its importance, for in cases of defective vision associated with intracranial disease there is nearly always change visible with the ophthalmoscope. Yet it is very important not to make too much of slight changes. Imperfect observation may detect "congestion" and "anæmia" of the disk when physiological redness and pallor are the true conditions. "Some observers," he says, "are really inferring when they should be only looking." He also pointed out that those who do not use the ophthalmoscope are apt to miss important changes and perhaps to misinterpret symptoms. It must be remembered that it is exceedingly common for a patient with intense optic neuritis to see quite well, and if such a patient died and a cerebral tumour were found, unless an ophthalmoscopic examination had been made the presence of inflamed disks would have been undiscovered, and the significance of such a condition missed. An illustration of the converse of this was the case of a patient who became almost blind and died. Post mortem, a condition of disease of the optic thalamus was discovered. In such a case the blindness might have been ascribed to the disease in the optic thalamus—quite erroneously, had it not been discovered by the ophthalmoscope that in each retina there were the extensive changes of albuminuric retinitis.

Optic atrophy also, it is pointed out, is important in reference to examination with the ophthalmoscope. It often occurs in tabes dorsalis and in general paralysis of the insane. It may occur with or without pupil changes, with or without pain, with or without ataxy. And, of course, it is not a necessary sign in tabes dorsalis.

Another condition noted with the ophthalmoscope is retinal embolism. This may be associated with evidence of cerebral embolism, and observation with the ophthalmoscope is important, as otherwise the optic atrophy, the result of embolism of the central artery of the retina, might be mistakenly supposed to be the result of cerebral disease.

Similarly also, it is pointed out, the ophthalmoscope may render visible other tissue changes in the fundus, such as those resulting from tubercle and syphilis and from Bright's disease. But it has to be noted that the ophthalmoscopic appearances of Bright's disease are often closely simulated by those occurring in intracranial tumour, while conditions really depending upon Bright's disease are often such as strongly to suggest the condition of the fundus occurring with intracranial tumour. Reference also was made to the visible arterial pulse in the fundus found in association with aortic regurgitation and to the retinal haemorrhages occurring in some cases of purpura.

In the paper [13] "Remarks on the Routine Use of the Ophthalmoscope in Cerebral Disease" he returns to a subject on which he had written early and often. He makes out a strong argument in favour of this procedure, and it may be said that it is chiefly due to his insistent teaching that the ophthalmoscope is now in such constant use in the hands of neurologists.

He quotes physicians and ophthalmic surgeons in support of his repeated allegation that marked optic neuritis may be present with perfect vision. Numerous cases are cited in which marked optic neuritis was present, yet no change was discoverable either in the acuity of vision or the extent of the fields. Yet in many of these cases post-mortem examination revealed the presence of intracranial tumour or the subsequent progress resulted in blindness. The practical importance of the discovery of optic neuritis is pointed out not only with reference to prognosis but also in regard to treatment, and it is again pointed out that neuritis may coexist with good vision and may pass off, leaving sight good.

Jackson published an important paper [28] on eye symptoms and tabes. It may be said that nearly all he tells us on the subject is now commonplace knowledge, but at the time it was written the paper set out points not then clearly grasped. He mentions three non-ocular signs in tabes—lightning pains, loss of knee-jerks, and ataxic gait—and three ocular—paralysis of oculo-motor nerves, pupil changes, especially the Argyll-Robertson phenomenon, and optic atrophy—whilst he clearly indicated that not all of these occurred simultaneously in many cases of tabes, but that two or three of them not infrequently occurred in the same case. But the important point was the recognition of any one of them in a patient as indicating the likely nature of the case and as suggesting the possible presence of others if carefully looked for.

In the same volume [28] there is the record of a very interesting discussion on "optic neuritis in intracranial disease" in which Dr. Jackson took part. His contribution is full of facts, and has many suggestive questions as well as statements. It is in six sections. The first deals with optic neuritis ophthalmoscopically. He says he used arbitrarily to make two stages in optic neuritis: (1) slight œdematous swelling, and (2) a climax of extensive great swelling, with hæmorrhages and strangulation of vessels, but he avers his belief that there is but one kind of optic neuritis from intracranial disease. But he points out again the simulation of the optic neuritis of intracranial disease by the condition found in Bright's disease, and mentions a case in which he himself had made the diagnosis of brain disease simply from the appearance of the fundus oculi, yet post mortem no such disease was found, but the patient, a young boy, was found to have granular kidneys. He asks, Can the fundus condition in such cases, as well as in cases of meningitis, be definitely distinguished from that in cases of intracranial tumour?

The next section (2) deals with clinical facts. Uniocular optic neuritis rarely occurs in cases of intracranial tumour. When it does, what is its significance? Does it ever result from cerebellar disease? He says he believes the diagnostic value of optic neuritis is not different whether sight be good, defective or bad. But physicians see more cases in which the vision is good than with even defect of sight. He also refers to the occasional temporary failure of sight and the rapid permanent failure of sight, which occur in some cases, and inquires as to their significance.

The third section is devoted to a consideration of optic neuritis with other symptoms, non-localizing, such as vomiting, headache, &c., and localizing, such as unilateral convulsions, slowly progressive unilateral weakness of limbs, head enlargement, sudden hemiplegia, as from hæmorrhage into a tumour, reeling gait in cerebellar cases, and cranial nerve palsies, and he points out that optic neuritis does not occur in ordinary epilepsy, with complete aphasia, or with extreme mental conditions.

Section 4 deals with the diagnostic and non-diagnostic value of optic neuritis. It usually occurs with intracranial tumour or some other adventitious product. The mass may be in any part of the encephalon, although probably tumour in the medulla oblongata rarely produces optic neuritis. Yet it may be absent with tumour in almost any part, i.e., "optic neuritis occurs with tumour in almost any part

of the encephalon, and may not occur with tumour in many parts of it." Also it may occur late in cases of cerebral tumour, or it may appear late and pass off, the patient dying ultimately of tumour. It is rarely found with softening from clot, it is not caused by mere destruction of any part of the encephalon, and, as has been insisted on before, it may be unattended with impairment of sight. Optic neuritis points to the general nature of the local disease, not to its particular nature. It means, as a rule, "foreign body," and, so far as the optic neuritis is concerned, that produced by a syphilitic or gliomatous "lump" is not to be distinguished from that produced by any other kind of foreign body.

It is important to distinguish between the development of neuritis from a foreign body and loss of sight from a destructive lesion. Thus hemiopia is usually the result of a destructive lesion and in itself implies no change in the fundus. Of course, a tumour so situated as to cause hemiopia will probably cause also optic neuritis. Yet in the ordinary hemiopia from a hæmorrhage or clot, fundal changes do not occur. "The several different lines of evidence agree in converging to the conclusion that optic neuritis in cases of intracranial adventitious products is to be looked upon as resulting secondarily from such products in their general, or more correctly in their abstract, character —that of 'foreign bodies.'"

Section 5 deals with the various hypotheses as to the mode of production of optic neuritis.

(1) von Graefe's idea that it is produced by raised intracranial pressure inducing venous congestion in the central vessels of the optic nerve. Against this is the fact that a small tumour may cause intense optic neuritis and that a large hæmorrhage may not cause, does not cause, optic neuritis.

(2) Hypothesis of Schmidt, which ascribes optic neuritis to distension of the optic nerve sheaths.

(3) Hypothesis of Benedict, first hinted at by Schneller, viz., that of reflex vasomotor action (analogy of convulsion) by induced instability in grey matter, and action in influencing vessels in optic nerves. Loss of sight, temporary and sudden, does occur with optic neuritis, and often occurs also at onset of a convulsion. Secondary changes in the brain are certainly produced by tumour as is shown by epileptiform seizures. Optic neuritis is thus supposed to be a doubly indirect result of tumour. The sequence is supposed to be: tumour, changes of instability, effects produced by the latter on muscular-walled arteries

of optic nerves and centres. Jackson considers this hypothesis, the vasomotor one, *the most plausible.*

The sixth section deals with treatment. Optic neuritis, although, secondary to tumour, becomes independent of tumour, becomes autonomous. He supposes that if we were able to reverse all central changes which had led to optic neuritis the optic neuritis would remain for treatment. He thinks it clear that the neuritis does become autonomous because atrophy which is permanent ensues when all other signs of cerebral disease are gone, when this disease has become quiescent. The best time for treatment is early. Hence the importance of early recognition by the use of the ophthalmoscope.

Jackson reported a case [29] of ocular movements produced by pressure on a diseased ear. He had at various times written on Ménière's disease and on the vertigo occurring in that condition. Vertigo, indeed, was a symptom which always excited his interest, whether it was ocular or auditory in origin, or if it were associated with epilepsy.

The patient was a woman with old-standing otorrhœa, in whom pressure on the right tragus produced movements of eyes, slow, to the *left*, followed by quicker movement back to the *right*. Objects appeared to move to *left*, i.e., with slower jerks. When she improved and jerks to *right* became slower, she was able to see objects move also to right.

He supposes that pressure on the tragus was transmitted by the chain of bones to the contents of the semicircular canals, and changes in the canals were propagated through cerebrum or cerebellum—possibly both—to ocular muscles. The case is also important as showing dependence of vertigo on ear disease. She was subject to giddiness and even occasional reeling.

His Bowman Lecture was delivered before the Ophthalmological Society on November 13, 1885. It is a philosophical treatise on Spencerian lines, but with definite practical application. Differentiation or division of labour, he says, is a universal law, and the body medical is, of course, subject to this law. Specialism is therefore a natural outcome, but specialists have to justify their differentiation. But the modern doctrine of evolution involves more than differentiation. The other factors are increasing definiteness, increasing integration, and increasing co-operation. The ophthalmic surgeon has justified himself in the factor of definiteness by his work on ocular muscles and errors of refraction; he has also justified himself in the factor of

integration. Thus Argyll-Robertson in the pupil symptom has given to neurologists not merely a new symptom but a means of investigation of several important diseases; and neurologists like Westphal and Erb have paid back the debt by showing the diagnostic value of the knee-jerk. The necessity of the fourth factor—co-operation—is shown, e.g., in such a case as one of hypermetropic headaches. The neurologist may be consulted for the headache, and he ought to be able to suspect hypermetropia as a cause. But only the skilled ophthalmic surgeon can estimate it precisely and correct it accurately.

The complexity of some nervous diseases is, he says, often underrated. Epileptic paroxysms and their after-conditions are an illustration of such complexity. When its symptomatology is considered, there is shown the necessity of different, definite, wide knowledge and co-operative work. Ophthalmic surgeons, aural surgeons, alienists and psychologists can all contribute to our knowledge of the symptomatology of the disease. Thus the ophthalmic surgeon may investigate the fundus. He may study visual aura—colour warnings or "seeing faces"; the condition of the pupil will be observed. Thus in uræmic convulsions pupils may be dilated and become smaller after the paroxysm. Vertigo also will have to be investigated by the ophthalmic surgeon as well as the aural. The condition of the eyes after a paroxysm also will be observed. Beevor found they were deviated to the side opposite to that to which they deviated at the commencement of an attack. Another curious eye symptom in the epileptic aura will demand attention, viz., the apparent alteration in the size and distance of objects, comparable with the micropsia after the instillation of atropine. Such are some of the phenomena requiring investigation in epilepsy at the hands of an ophthalmic surgeon.

But there are other non-ophthalmological symptoms of epileptic paroxysms, especially of those of organic parts. These are an important part of the evidence, showing that the "organ of mind" (the highest centres) represents all parts of the body. The highest centres are the organs of will, memory, reason and emotion, the four elements of mind, i.e., of consciousness. "No wonder the epileptic discharge, beginning in some part of the 'organ of mind,' produces universal bodily effects, animal and organic. Unless retinal impressions corresponding to colour of objects, and ocular movements corresponding to shape of objects, be represented in the highest centres, how are we to account for the physical basis of visual perception and ideation? If the organic parts are not represented in the highest centres, an emotional manifestation,

say of fear, is unaccountable, and so, too, are the physical symptoms of some cases of melancholia, the insanity of fear (anxiety, depression, &c., being only 'fear spread out thin.')"

Much attention is given to the movements of chewing or tasting, swallowing, &c., in association with slight fits, and especially associated with the "dreamy state." He expresses the belief that they, as well as another phenomenon occurring in epileptic warning—the apparent alteration in size or distance of external objects—are the indirect (reflex) results of epileptic discharges of sensory elements. He also believes the "dreamy state," "seeing faces," "hearing voices," and such elaborate mental states arise during slightly raised discharges of healthy nervous arrangements, i.e., arrangements untouched by the epileptic discharge. The "dreamy state" he regards as a very voluminous mental state—not an aura, although it has been called an "intellectual aura"—and he believes that the subjective sensations—smell, taste (chewing movements, &c.), and the epigastric sensations—most often occur in cases of epilepsy in which there is a dreamy state, and depend upon lesions in the cortical area supplied by the posterior cerebral artery. When the warnings are of colours or sounds, the lesion is probably in the cortical area of the middle cerebral, and he enunciates his belief that the nervous changes (discharging lesion) in epilepsy are not primarily but only secondarily nervous; that in most cases they are secondary to embolism or thrombosis of small arterial branches.

Attention is next directed to the after-condition of epileptic paroxysms. Insanity may occur in at least two forms—post-epileptic "unconsciousness" with mania and acute dementia (coma). Physically this is universal paralysis, and paralytic phenomena are present, such as transitory lateral deviation of the eyes, exaggerated knee-jerks, and ankle clonus.

For the study of such a complex disease, therefore, the neurologist must avail himself of the work done by different specialists. "We require many different kinds of *definite* or technical knowledge. All the sagacity in the world will not suffice either for practical ends or for the scientific investigations of complex problems without technical knowledge."

The only method of investigating such a complex disease is that by the use of hypothesis, and it is suggested that the hypothesis of evolution should be adopted for this purpose. According to this all parts of the body are represented in each of three levels of evolution.

"Representation increases in differentiation, definiteness, integration and co-operation from lowest centres to highest centres. The 'organ of mind' is nothing else than a series of centres representing, or what is the same thing co-ordinating, all parts of the body 'from nose to feet' in greatest complexity. The evolutionist does not attempt the marvellous feat of 'getting the mind out of the body,'" he only tries to get the physical basis of mind (highest centres) out of the rest of the body.

To test this evolutionary hypothesis one should use the experiments which disease makes in effecting dissolution. Thus paralysis of the ocular muscles from affection of their nerve trunks is an example of disease of the lowest level. So are ophthalmoplegia externa and interna. Lateral deviation of the eyes occurs in lesions of the middle motor level, and deviation of the eyes in and after epileptic fits is probably the result of lesions in the physical bases of innumerable visual ideas and other mental states.

It is also suggested that we may have atrophies of cells to organic parts analogous to atrophies of anterior horn-cells. Thus diabetes may be the result of a nuclear atrophy of cells of that part of the great vasomotor centre which especially governs the hepatic artery. Similarly Graves's disease and myxœdema may be of similar etiology. These, of course, we regard as being lesions of the lowest evolutionary level. But these examples must be compared and contrasted with the symptoms of negative lesions of the highest level. After an epileptic fit the condition is one of nearly universal paralysis (dissolution effected).

"Since the epileptic discharge starting in the highest level produces effects in nearly all peripheral parts, currents from this primary discharge must have traversed and discharged more or less of the middle and lowest centres in order to get at the periphery. Hence the paralytic condition after a severe fit will be a very compound one, all orders of centres being somewhat affected, but in different degrees."

In conclusion, it is urged that the neurologist, by availing himself of the work of ophthalmic, laryngeal, aural, &c., surgeons along the lowest line of evolution, by working himself at the middle, by co-operating with alienists in the study of diseases of the highest level, may hope to justify his specialism. And the different workers may hope to add different knowledge, to attain to more definite knowledge, to further the integration of general medical knowledge, and to lead to higher and more methodical co-operation of different workers.

In 1886 there was a discussion [30] at the Ophthalmological Society

on Graves's disease. Jackson's contribution was brief but important. He said that he attached much importance to von Graefe's sign, although he had seen well-marked cases of Graves's disease in which it did not occur, mentioning in illustration two cases in sisters. He suggested a comparative study of such cases with some other family diseases. He mentioned that in the recent cases of Graves's disease he had seen, eight in number, the right lobe of the thyroid was the larger, a fact bearing on the question of the central pathology, as W. A. Fitzgerald had suggested, for the right vagus had, in some lower animals, more inhibitory influence on the heart than the left. He also expressed the opinion that the hypothesis of a central pathology was the most probable, and referred to the experiments of Brown-Séquard and Filehne on the production of exophthalmos by injuries of the restiform body. In all cases of fatal Graves's disease the medulla oblongata and pons Varolii should be carefully examined microscopically. He referred to a case mentioned by Warner and Bristowe in which there was *ophthalmoplegia externa*, and a case which he had seen complicated with asthmatic paroxysms, and one with paroxysms of right facial spasm. Pavy had noted the association of Graves's disease with diabetes. In the so-called complicated cases the association might be accidental, but they deserved investigation. He did not remember seeing Graves's disease in a man.

In his Presidential address [31] to the Ophthalmological Society, Jackson says that no department of medicine had greater attraction for him than ophthalmology. It was the first subject at which he specially worked after his student life, and he gratefully acknowledges—as he so often does in his writings—his indebtedness to the example and teaching of Jonathan Hutchinson while he was his clinical assistant at Moorfields. He emphasizes the benefits of the opportunities for being well disciplined in exact observation which an ophthalmic hospital affords, and the effect of such discipline in teaching students to avoid vague and indefinite statements.

"Since six cranial nerves and the sympathetic nerve," he goes on to say, "supply the eyeball and its apparatus, it is evident that without a good knowledge of eye diseases the thorough investigation of very many morbid affections of the nervous system is not to be methodically carried out. . . . Unless the physician uses the ophthalmoscope by routine he will often enough overlook the best evidence—and I am convinced in many cases the only decisive evidence—of gross organic disease of the brain there is to be had; and if, as is most often the case

in a physician's practice, sight be good, he will not surmise that there is anything wrong with his patient's optic nerves, and will very likely be incredulous when someone who has looked at them tells him that there is swelling of the disk." Further, without special knowledge, paralysis of the superior oblique may be overlooked, and the symptom—vertigo—for which the patient seeks advice may be misinterpreted. So also hypermetropia as a cause of headache may be overlooked and the hypermetropic disk, unless one is familiar with its appearance, may be regarded as a condition of optic neuritis indicating serious brain disease. So that "without a good knowledge of ophthalmology a methodical investigation of diseases of the nervous system is not merely difficult but impossible."

He also suggests that cases of uniocular optic neuritis with tumour of one hemisphere are the cases which will throw most light on the process by which optic neuritis results from disease in various parts of the encephalon. "At present," he says, "there are only hypotheses as to this process," although he regards the hypothesis of vasomotor action as the "most plausible." Various other conditions have to be accounted for: the epileptiform paroxysms, the sudden temporary loss of sight, the vomiting and slowing of the pulse, the tendency to sudden death, and the question is asked, Do these symptoms not dependent upon optic neuritis depend upon a similar morbid condition in other nerves, such as the pneumogastric and its centres, as suggested by Thomas Buzzard?

The integration of ophthalmological knowledge may also help, by the study of nystagmus, to elucidate the tremor of Graves's disease and the various occupation spasms such as writer's cramp, and the work of the ophthalmic surgeon in ocular paralysis may help to the interpretation of inco-ordination produced by destructive lesions of nervous centres in animals.

Further, in the study of eye conditions we are most likely to be able to trace the ascending complexity in the evolution of movement from ocular muscles to their representation in most complex movements in the highest motor centres, i.e., in the physical bases of visual ideas. Such a study would do much towards showing that the organ of mind is sensorimotor, and we should then place the study of epilepsy proper and insanity on a realistic basis. Further, the great importance of a study of ocular paralysis is insisted upon in reference to the question whether mental states do or do not occur with the "outgoing" currents as well as with the "ingoing" current, and this

question should interest alienist physicians in ophthalmological work. And physicians and aural surgeons especially can help ophthalmology by showing and investigating cases such as spasmus nutans with nystagmus; tetanus, as to whether ocular muscles are implicated in this disease; and cases of auditory vertigo, especially if associated with ocular movements. He concludes with a quotation from a paper by James Anderson [20]: "It seems to me the best and most hopeful feature of ophthalmology that it has relations, closer, or more remote, with every branch of medicine and surgery, indeed with almost every branch of science."

In the introductory part of this address I referred to the series of "Neurological Fragments" published by Dr. Jackson. The first [6] of those deals with a very interesting condition. Mendel [19], as a result of a series of experiments on animals, formulated the hypothesis that the oculo-facial group of muscles (frontalis, corrugator supercilii and orbicularis palpebrarum) is really innervated from the third nucleus, although the fibres to these muscles are distributed in the seventh. A case published by Tooth and Aldren Turner [3] lent support to this view. In the first neurological fragment Dr. Jackson considers the same point, and gives an account of the investigation of two cases of ocular palsy with ptosis in which weakness of the orbicularis could be demonstrated. He alludes to the fact mentioned by Duchenne that the orbicularis palpebrarum really consists of four muscles, and to the fact mentioned by Fuchs that with regard to closure of the lids we have to distinguish between moderate closure, as in winking and sleeping, and the process of screwing the lids tightly together. No doubt, Jackson says, the muscle is represented in several places in the central nervous system according as it takes a share in different movements. He suggests that one of these places for the representation of the orbicularis palpebrarum is that in which the external ocular movements for directing the eyes for the estimation of distance are represented. Presumably the peering movements of a short-sighted person, movements helping him to see better, will be represented along with certain movements of the eyeballs, and as the third nerve is that chiefly implicated in ocular movements the representation of the orbicularis so far as these movements are concerned will be most probably in the nucleus of the third nerve.

In the second neurological fragment [7] he refers to the interesting class of cases in which with congenital ptosis elevation of the eyelids can be produced by masticatory movements, but especially by

lateral movements of the jaws produced by the external pterygoid muscles, a fact suggesting, as was pointed out by a committee which investigated Marcus Gunn's case of this character, that in such cases the levator palpebrarum is supplied both from the nucleus of the third nerve and also from the external pterygoid portions of the nucleus of the fifth nerve. "It seems pretty clear," he says, "that the point of emergence of a nerve trunk from the central nervous system is a very untrustworthy clue to the nuclear origin of the several fibres making it up."

In the third neurological fragment [9] suggestions are made as to the use of cocaine for the investigation of abnormal conditions of the eyes, taking advantage especially of its characteristic, that it stimulates the endings of the sympathetic, dilating the pupil and widening the ocular aperture. He suggests, e.g., observations on the effect of cocaine on the Argyll-Robertson pupil and on the conditions of suddenly occurring immobility, under all conditions, of one pupil, with dilatation and loss of accommodation of the same eye, and on the reaction to cocaine of the pupil at different ages. It should also be used, he suggests, in cases of congenital ptosis, in order to ascertain, by its effects on Müller's muscle (stimulation of which in the normal condition widens the ocular aperture), whether that is affected in those cases as well as the levator palpebrarum.

The fourth neurological fragment [10] deals with the condition of the pupil and eyelids in cases of paralysis of the cervical sympathetic. Long ago [12], he mentions, Jonathan Hutchinson drew attention to the peculiarities of ocular paralysis from lesion of the cervical sympathetic. In cases of fracture dislocation of the cervical spine the pupils may be of equal size and may contract to light and during accommodation, yet if the investigation is not pushed further a very interesting sign may be missed. After speaking of paralysis of the "dilating" fibres of the pupil in such cases, Mr. Hutchinson wrote: "This is a very important and valuable symptom. It occurs only when the injury is in the cervical or upper dorsal region. The pupil is neither dilated nor much contracted. It is simply unable to dilate. Unless carefully examined the myosis, being so slight in degree, may easily be overlooked. The plan is to examine the eyes in shade, and you will then find that the pupils remain just of the same size as they were when exposed to light. Sometimes one pupil is more definitely contracted than the other." He mentions a case of a wound of the neck under the care of the late Mr. Marcus Beck, in which only one eye was thus affected. The pupil

of the affected eye did not dilate, as did that of the other eye, when the patient's back was turned to the window, nor did it dilate on faradic excitation of the skin, and Dr. Head found that cocaine had no action on the pupil or on the lids of the faulty eye. Cases of rupture of the brachial plexus, cases of thoracic aneurysm, cases of wounds and tumours of the neck, should be investigated in reference to this point, and so also should cases in which the small muscles of the hand are affected, especially cases of syringomyelia and of anterior poliomyelitis. He had referred in the Bowman Lecture to a case which he supposed to be a case of progressive muscular atrophy in which there was affection of one pupil, believed to betoken paralysis of so-called dilator fibres given off by the cervical sympathetic nerve to the iris and to Müller's muscle, a case which he now, by reference to the original notes, had little doubt was one of syringomyelia. He had been puzzled by finding no such pupil abnormality in undoubted cases of the Aran-Duchenne type of progressive muscular atrophy. He suggests that fibres ultimately passing in the cervical sympathetic to palpebro-ocular structures may not go through anterior horns, and if they do they may not atrophy in Aran-Duchenne progressive muscular atrophy. The effect of cocaine in such cases and in cases of anterior poliomyelitis, the hands being affected, may throw light on the exact course of the fibres in question.

In a paper, the last which he published [11] "On Some Abnormalities of Ocular Movements" written in association with Leslie Paton, a series of very interesting observations and experiments is described, undertaken with the view of elucidating whether "erroneous projection" which occurs in paralysis of, e.g., an external rectus muscle is the result of an increased outgoing current, as held by Bain and Wundt, or of an ingoing current as suggested by Müller, and a little later by James.

He remarks that movements of both eyes are represented in each half of the brain, but the lateral movements of both eyes to one or other side are most specially represented. But as he had remarked [8]: "Every movement called voluntary will be represented in the cerebral hemispheres, and thus there must be representation in them of all ocular movements a man can effect when he tries." "I asked Dr. Risien Russell," he says, "to make researches on the matter. At my suggestion he cut the right external rectus and the left internal rectus of a monkey, thus rendering movements of the two eyes to the right impossible. Upon faradic excitation of the animal's left cortical area, the eyes turned directly upwards or directly downwards according to

the particular part of the eye area stimulated" [4]. This suggestion of Jackson's has always seemed to me one of the simplest yet most illuminating which he ever made.

The basis of the experiments carried out by Paton was the artificial simulation of paralysis of an external rectus muscle by an appropriate prism. So far as secondary deviation and erroneous projection are concerned there will be no material difference between prism paralysis and ordinary external rectus paralysis. But it is obvious that as all the nervous tracts, ingoing and outgoing, remain intact, such an experiment can offer no support either to the outgoing or ingoing theory. But an interesting and almost crucial observation was made on a man who had paralysis of his left third nerve, paralysis of the right sixth and paresis of the right third nerve, upward, downward and inward movements being defective. The varied observations on erroneous projection need not be detailed, but the result of these, in the words of the observer, "seemed to be definitely opposed to the hypothesis that the false projection in the case of one eye where one of the muscles is paralysed may be due to impulses coming up from the muscles of the sound eye," for as soon as one of the paralysed muscles of the right eye was called into action the patient got false projection in the direction of action of the respective muscles. Such an effect in this case could not be the result of afferent impulses from the other (the left) eye, which was covered, since the left eye was always in a position of left deviation.

It is not easy to sum up briefly the outcome of Jackson's teaching in the various papers to which reference has been made. From the practical point of view his insistence upon the routine use of the ophthalmoscope is to be noted because of the importance of its revelations in reference to diagnosis, prognosis, and treatment, and because of the scientific bearing of its discoveries on the symptomatology of intracranial and other diseases. Optic neuritis itself, he asserts, has no localizing value, but he says that the study of instances of uniocular optic neuritis and the careful examination by every means in our power of such cases may furnish us with a clue to the discovery of the real nature of optic neuritis. It is interesting to know that at one time, impressed apparently by the association of speech defect with lesions of certain parts of the left hemisphere, he thought that defects of sight might be similarly associated with disease in a part of the right hemisphere [24] :—

"I have been struck with the greater frequency," he says, "with

which I have found optic neuritis with hemiplegia on the left than on the right side. The facts I have observed go to prove that disease in a certain region of the left hemisphere produces loss of speech, and other facts go to show that disease in the corresponding part on the right does not affect speech at all. Having found disease here on the right with amaurosis several times, I have been led to pay much attention to this part, and I was at one time sliding into the belief that disease of it might be more likely to induce amaurosis than disease of the other parts of the right hemisphere."

A second point which emerges from a consideration of these papers is the importance he attached to ocular palsies in relation to vertigo. The study of this connected directly both with epilepsy and locomotor ataxy, and also with labyrinthine disturbances, but he dwelt on its importance in a wider sense, viz., because the facts of ocular vertigo are of great value in supporting from analogy the dictum that the anatomical substratum of a word is a motor, an articulatory process.

A third point which is to be noted is the remarkably high opinion which Jackson entertained of the educative effect of work carefully and energetically pursued at an ophthalmic hospital. He himself, as he repeatedly acknowledges, derived the greatest help from his early work at Moorfields, both because of the educative effect of the experience and the discipline in exact observation which it imposed. Such work, however, need not lead to a mere barren specialism or differentiation. As a matter of fact, it does not, for while ophthalmic studies have resulted in the increase in definite knowledge of eye conditions and diseases, they have also led to discoveries which have been a means of investigation of several important diseases, and they have been productive of the co-operation of neurologists and ophthalmic surgeons in the study and treatment of different morbid conditions. Such considerations obviously embody the evolutionary idea, and have in them the germ of the philosophical treatises on evolution and dissolution of the nervous system to which I have briefly referred.

I feel that I have not done justice to my subject. I doubt whether I could, if I were to elaborate it at much greater length. But I end on the note on which I began, for I trust that what I have said may open to some a new book, and may be to others a reintroduction to an old acquaintance. To me the study of these papers has been a source of intense interest and of great pleasure, and I have been anew impressed with the marvellous powers of one who was to many of us here a constant stimulus, and was besides, to me, a very dear friend.

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THE SENSORY FIELD OF THE FACIAL NERVE: A FURTHER CONTRIBUTION TO THE SYMPTOMATOLOGY OF THE GENICULATE GANGLION.[1]

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CHAPTER I.—INTRODUCTION.

AT a meeting of the American Neurological Association in May, 1907, a preliminary statement was made of my views regarding the general sensory system of the facial nerve and its clinical importance. This subject was still further elaborated in my presidential address to the New York Neurological Society in February, 1909 [18], and the attempt was made to outline the sensory system of the seventh cranial nerve, exclusive of its taste function and muscle sensibility, and to endow this system with a definite symptomatology similar to that of the other mixed cranial nerves. Thus the geniculate ganglion, its posterior root, the pars intermedia of Wrisberg and various peripheral divisions, were accorded a definite place in the realm of clinical neurology, supplementing the position which this system had long occupied morphologically in the domain of embryology and comparative anatomy (see fig. 1).

[1] Amplification of a subject presented at the Seventh Annual Meeting of the American Society for the Advancement of Clinical Investigation, held in Washington, May 10, 1915.

The sensory system of the geniculate ganglion in man stands in close relation to the structures of the auditory mechanism, a natural sequence of its course and distribution in the lower vertebrate forms where it is the nerve of the first branchial or ear cleft. It participates

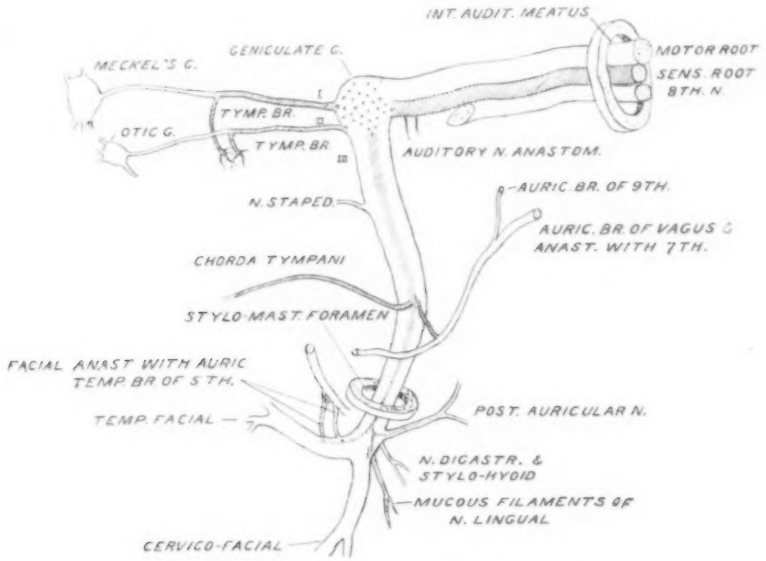

FIG. 1.—Diagrammatic representation of the facial nerve, showing the geniculate ganglion, its sensory root (the nerve of Wrisberg) and peripheral divisions.

1st Division—Great superficial petrosal nerve passing to Meckel's ganglion and its tympanic branch (great deep petrosal).

2nd Division—Small superficial petrosal nerve passing to the otic ganglion and its tympanic branch (small, deep petrosal).

3rd Division—The motor trunk, including the chorda tympani and sensory fibres to the external ear.

The sensory filaments in the motor trunk may reach the auricle by way of the auricular branch of the vagus; the posterior auricular nerve, by anastomoses of the temporo-facial with the auriculo-temporal branch of the fifth, or with the motor fibres destined for the minute intrinsic muscles of the external ear.

The lingual branch of the facial conveys mucous filaments to the anterior pillar of the fauces and adjacent palatal region.

in the innervation of the internal ear, the middle ear and its prolongation into the mastoid cells and Eustachian tube, as well as the skin of the auricle. There is also evidence to show the occasional presence of a vestigial innervation within the buccal cavity, on the palate and in

the chorda distribution of the tongue, a remnant of the large intra-oral system which exists in fishes.

Among the more important symptoms which I referred to this sensory system are:—

(1) Various clinical forms of the geniculate neuralgia, viz., primary tic douloureux of the ear; reflex and post-herpetic otalgia; tabetic otalgia associated with degeneration of the nerve of Wrisberg.

(2) Auditory and mastoid pains in cases of facial palsy (Fallopian neuritis), and in recent cases the occasional presence of hypæsthesia in the region of the concha auris.

(3) As a sensory pathway for the direct transmission of irritative stimuli to the facial nucleus, thus favouring the production of nuclear irritability with resultant facial twitchings, myokymia or spasm.

(4) Herpetic inflammations of the geniculate ganglion; a *syndrome* characterized by an eruption of herpes zoster on the central portions of the external ear, usually associated with facial palsy and frequently with auditory nerve symptoms: the acoustic disturbances when present varying in intensity from slight hypo-acousis to the severer forms of Ménière's disease.

In a later study [21] the special symptomatology of herpetic inflammations of the auditory, glosso-pharyngeal and vagal ganglia was also considered, and a provisional attempt was made to outline the various zoster zones of these ganglia both on the external ear and within the buccal cavity.

My object in approaching this field again is to review briefly some of the contributions which have been made by other observers since attention was first directed to the subject; to answer certain criticisms which have been directed against it, and to present the results of subsequent personal study and observations, more especially those pertaining to the cutaneous and intra-oral fields of the seventh nerve.

Chapter II.—A Review of Recent Contributions to the Sensory Functions of the Facial Nerve.

(A) *The geniculate neuralgia.*—The most important of the recent contributions to the symptomatology of the geniculate system is that by Clark and Taylor [2], in which a case of severe tic douloureux of the sensory system of the facial nerve was cured by intracranial section of its posterior root, the pars intermedia of Wrisberg. The pains were typically neuralgic in character, paroxysmal, lancinating, and very severe, and were localized chiefly in the depth of the ear, the anterior

wall of the external meatus, and on a small area just in front of the ear (otalgia). Immediate and complete relief was obtained after section of the posterior root, the so-called physiological extirpation of the ganglion, a method advocated by Van Gehuchten and Spiller in cases of trigeminal neuralgia. This patient had been examined by a number of neurologists and all were agreed as to the true neuralgic character of the pain, and its limitations to the zone which I had outlined for the geniculate otalgia. Allen Starr [42], who had examined the patient independently, also reached the same conclusions and had advocated surgical interference similar to that which was carried out subsequently by Clark and Taylor. Through the courtesy of Dr. Clark and Dr. Taylor, I had several opportunities of examining this patient, and in my opinion the case was one of true geniculate neuralgia. Furthermore, it is of interest to record that the patient had been under observation at various times since the operation and only recently—i.e., six years after the root section—she was seen by Dr. George Crile, of Cleveland, who reported complete freedom from neuralgic pain in the ear. There has never at any time been a recurrence of the otalgia. One would, therefore, seem justified in accepting a case of this character and the brilliant result achieved by operation as a complete confirmation of views which I had previously expressed as to the sensory system of the seventh nerve, its relation to otalgia, the surgical possibilities offered, and that the geniculate neuralgia merits a definite place in the large clinical group of this affection [20 and 23].

Cases of a similar nature have apparently been encountered by Wilfred Harris [15] in his extensive experience with the treatment of trigeminal neuralgia by the method of alcohol injections. His comments on the subject of geniculate neuralgia are as follows: "Excluding those cases, not very uncommon, in which some neuralgia in front of the ear accompanies facial palsy or herpes within the auditory meatus, possibly due to geniculate inflammation, I have encountered two patients who suffered from almost identical paroxysmal neuralgia, which had lasted for ten and eleven years respectively, and in which the paroxysms of pain began with the throat or posterior palatal region on one side, and spread into the ear and in front of the ear on to the cheek, and down the side of the neck. The sudden onset and intensity of the pain and the recurrent paroxysms closely resemble chronic trigeminal neuralgia, but the distribution of the pain made me doubtful as to dependence on the fifth nerve, and in each case no alleviation was produced by successful injection of the third division of the fifth."

Charles K. Mills [30], in a critical discussion of the sensory functions attributed to the seventh nerve, opposes some of the views which I had advanced, and is not inclined to accept Clark and Taylor's case as one of pure geniculate neuralgia. He offers the suggestion that the pains may have been merely symptomatic and relieved by the decompressive effects of the operation, and not by section of the sensory root of the seventh. He would eliminate the case on these grounds as confirmatory of my views. It should be remembered, however, that this patient was observed not by one, but by a number of experienced neurologists (Allen Starr, Pierce Clark, Leszynsky, and Hunt), all of whom testify to the typical and strictly neuralgic character of the pain; the suggestion, therefore, that simple decompression could have affected a permanent cure in a case of this character—*intractable tic douloureux*—is, to put it mildly, a forced and unlikely hypothesis.

(B) *The sensory system of the seventh nerve and its relation to reflex facial twitchings and spasms.*—That the sensory system of the seventh nerve may be an important pathway by which irritating stimuli pass to the facial nucleus was emphasized in my original discussion of the symptomatology of this system. In this connection the Clark and Taylor case of geniculate neuralgia, already cited, is of considerable interest. At the presentation of this patient to the New York Neurological Society in May, 1910 [1], i.e., one year after the nerve had been divided, I called attention to the absence of the usual facial twitchings and spasms on the paralysed side. The patient had had originally a complete facial paralysis following division of the facial nerve, the division including its sensory root, the pars intermedia of Wrisberg. After the operation there had been gradual improvement and, at the time of the presentation, there was a very considerable return of power in the left side of the face, so that the eyelid could be closed and the angle of the mouth drawn to one side, and yet there was a complete absence of the contracture, with twitchings and over-action, which usually complicate the severer types of peripheral facial palsy. The absence of these motor phenomena in this case may be explained on the ground that the sensory root of the seventh had been divided, thus blocking the flow of afferent irritative stimuli to the nucleus. By this procedure the nucleus would be placed completely at rest as regards afferent stimuli through the facial system, and this may have had a bearing on the absence of the usual spasmodic manifestations. That section of a posterior root with consequent interruption of sensory stimuli may affect favourably spasmodic states and over-action of the

motor neurones has been amply demonstrated by the results of Foerster's operation in spastic paralysis.

Only recently Lasarew [29] has emphasized the concurrence of paresis in one branch with contracture or over-action in one of the other branches of the facial in lesions which involve the nerve in the posterior fossa, especially neuro-fibromata of the acoustic. This phenomenon of paresis associated with contracture, as well as facial twitchings and myokymic quiverings, I have frequently observed in cases of acoustic tumour, and have thought that irritation of the sensory root of the facial may have been in part responsible for some of these peculiar phenomena—a theory which is more in harmony with such motor manifestations than would be that of distant compression of the nucleus through the medulla, or irritation of the motor portion of the nerve.

(C) *Herpetic inflammations of the geniculate ganglion.*—Since the publication of my monograph in 1910 on the "Herpetic Inflammations of the Geniculate, Auditory, Glosso-pharyngeal and Vagal Ganglia" [21] there have been a number of contributions to this subject, among the more important of which may be mentioned those of Claude and Schaeffer [4], Dombrowski [11], Closier [6], Dabney [8], Norman Sharpe [39], Dejerine, Tinel and Heuyer [10]. Cases of this affection have also been reported by Climenko [5], Sharpe [38], Muck [32], Hennebert [16], Halphen [13], Palmer [34], Mollison [31], Laignel-Lavastine [27] and Souques [41], the symptomatology corresponding to one or other of the various clinical forms of this affection.

Of special interest is the case reported by Claude and Schaeffer with herpes occipito-collaris et oticus, because of its severity, the evidences of multiple ganglionic involvement, and the large number of paralytic complications, the latter including unilateral paralysis of the intrinsic muscles of the eye, the abducens, the facial and auditory nerves. A case very similar in severity and extent of distribution was reported many years ago by Raynaud [36].

The case described by Dejerine, Tinel and Heuyer is a typical example of the geniculate syndrome, but has a special interest because of the wide extent of the anæsthetic area and the interpretation which it receives from these authors. In addition to anæsthesia in the conchal region, there was also demonstrable a hypæsthesia of the face and of the occipital region on the paralysed side, all of which was referred to the geniculate sensory area. This subject will receive special consideration in Chapter V.

Charles K. Mills [30] and Kidd [25] have both contributed critical papers in which my views regarding the sensory functions of the facial nerve have been challenged. Mills upholds the old traditional point of view, and would limit the sensory symptomatology of this nerve to the special sense of taste. He does not admit the existence of herpes zoster oticus of geniculate origin, but would interpret the eruption in such cases as trigeminal or cervical herpes, or, possibly, of glosso-pharyngeal-vagal origin. He does, however, accept the possibility of an inflammation of the geniculate ganglion in herpes zoster, but this would produce merely disturbances of taste and facial palsy, without herpetic eruption, and if this were present it would indicate an involvement of one of the other ganglia, viz., the fifth, ninth, tenth, or upper cervical.

Kidd, on the other hand, admits the probable existence of a visceral sensory function of the seventh nerve, and confines his criticism to the cutaneous representation on the auricle, the existence of which in man he strenuously denies. The answer to many of the objections which are brought forth by these writers will be found in my earlier contributions. It is, however, my purpose in this paper to consider in detail some of the questions which have been raised, and more especially that of the cutaneous zone, and to formulate more definitely some of my views regarding this subject, fortified as they are by subsequent experience and by contributions from other workers in this field.

Chapter III.—The Cutaneous and Intra-oral Zones of the Geniculate Ganglion outlined by the Herpes Zoster Method.

In my discussion of the distribution of fibres originating in the cells of the geniculate ganglion, it was shown that this sensory system bears a very close relation to the structures of the auditory mechanism, its filaments passing to the internal ear, the middle ear and its prolongations, and to the external ear. I will now consider in detail the cutaneous innervation on the external ear, for, like the fifth, ninth, and tenth nerves, the seventh has not only an intra-oral, but a cutaneous representation as well. And as the geniculate sensory field has been the subject of some controversy, as well as misapprehension, I will first review briefly my own views on this question, and then proceed to the more recent contributions of other writers bearing upon this question.

In my previous studies the attempt was made to outline the

cutaneous zone of the facial nerve by the herpes zoster method, i.e., by the study and analysis of cases of pure herpes zoster oticus, in which the eruption was limited to small cutaneous areas on the external ear, corresponding in distribution to the geniculate, glosso-pharyngeal, or vagal ganglia. A detailed presentation of the various embryological, anatomical, and clinical facts which were gathered in support of this theory will be found in my earlier publications. For the present purpose it will be sufficient to quote certain of the conclusions which were reached in regard to the cutaneous zone of the seventh nerve [21].

"In eighteen of the cases (herpes oticus) the eruption was distributed in what I regard in part as the geniculate area on the external ear, i.e., the external meatus, concha, tragus, anti-tragus, anti-helix and fossa of the anti-helix, and the upper portion of the external surface of the lobule. This representation of the geniculate on the external ear is corroborated by the occasional presence of hypæsthesia in the region of the concha in cases of recent peripheral facial palsy. From the herpes zoster evidence I believe that we are justified in drawing the following conclusions. Pure herpes zoster oticus is dependent upon herpetic inflammation of the geniculate, glosso-pharyngeal and vagal ganglia. The ganglion of the tenth and the ganglion of the ninth are represented in part on the posterior portion of the tympanic membrane and auditory canal, as well as on the postero-mesial surface of the auricle and adjacent mastoid region. The zoster zone of the geniculate ganglion is represented on the external surface of the auricle, intercalated between the zone of the trigeminus in front and cervical ganglion behind. From the relation which the facial bears to the first visceral cleft, it is very probable that the zone of the geniculate also dips into the auditory canal as far as the tympanic membrane in the same manner as do the auditory strips of the trigeminus and glosso-pharyngeal vagus. As I have already indicated, a considerable allowance must be made in these zones, as I have outlined them for anatomical anomalies of various kinds and for the normal physiological variation and overlapping of sensory areas. Furthermore, these sensory areas are small, and, like the minute muscles of the external ear, must be regarded as more or less vestigial in character; in other words, as remnants of sensory zones which are gradually fading away under the overlap of larger and more important sensory systems."

The cutaneous zone of the facial nerve in this study was therefore localized on the central portions of the auricle, viz., the concha, the

external meatus, tragus and anti-tragus, incisura intertragica, anti-helix and fossa of the anti-helix, and the upper portion of the external surface of the lobule.

The cutaneous zones of the glosso-pharyngeal and vagal ganglia were localized on the posterior portion of the tympanic membrane, the posterior wall of the auditory canal, together with a strip on the postero-mesial surface of the auricle and the adjacent mastoid region. In addition to their cutaneous zones, it was also shown that the glosso-pharyngeal and vagal ganglia have intra-oral fields as well, the herpes zoster pharyngis and the herpes zoster laryngis, respectively. For embryological reasons, and also because of occasional herpetic manifestations, it was thought that the geniculate was also represented within the auditory canal and on the tympanic membrane, thus sharing with the fifth, ninth and tenth nerves in the innervation of this region.

In the description of these auricular zones of the seventh, ninth and tenth ganglia, especial emphasis was placed upon their vestigial character and variability. They were regarded as sensory remnants which in the course of phylogenetic development are being replaced by the encroachments of the trigeminal and cervical areas. These facts seem to have been overlooked by some observers, and notably by Kidd [25] in his discussion of the geniculate cutaneous area.

Cases of herpes oticus in the geniculate zone	Auricle	Concha	External meatus	Tragus	Anti-tragus	Anti-helix	Fossa of anti-helix	Lobule	Post-auricular cleft	Tympanum	Auditory canal	Soft palate	Facial palsy	Acoustic symptoms
Hennebert	..	+	..	..	..	..	..	..	+	..	..	..	+	+
Idem	+	..	+	..	..	..	..	..	..	..	+	..	..	..
Idem	..	..	..	+	..	..	..	..	..	..	..	..	+	+
Muck	..	+	..	..	..	..	..	..	..	+	+	..	+	+
Dejerine	..	+	..	..	..	+	..	+	..	..	..	..	+	+
Mollison	..	..	+	..	..	..	..	..	..	..	..	..	+	+
Palmer	..	+	..	..	..	+	+	..	..	..	+	+	+	+
Closier	..	+	+	+	..	..	..	..	..	+	+	+	+	+
Climenko	..	+	+	..	..	..	..	..	..	..	..	..	+	+
Sharpe	..	+	+	..	..	..	..	..	..	..	..	..	+	+
Idem	..	..	..	..	+	..	..	..	..	..	..	..	+	..
Idem	..	+	..	..	..	..	..	..	..	..	..	..	+	..
Personal observations ..	..	+	+	..	..	..	+	..	+	..	..	..	+	+
Idem	..	+	..	..	..	..	..	..	..	..	..	..	+	+
Idem	..	+	..	..	..	..	..	..	..	..	..	..	+	+
Idem	..	+	+	..	..	..	..	..	..	..	..	..	+	..
Idem	..	+	..	..	..	..	..	..	..	..	..	..	+	+

The distribution of the eruption is indicated on the anatomical landmarks of the ear by +.

Since these conclusions were reached in 1910, twenty additional cases of isolated herpes zoster oticus are available for analysis in

which the eruption was limited to certain small areas on the external ear, the trigeminal and cervical zones being free from eruptive manifestations. Of this number seventeen cases are to be regarded as occurring within the cutaneous geniculate zone. (See table, p. 426).

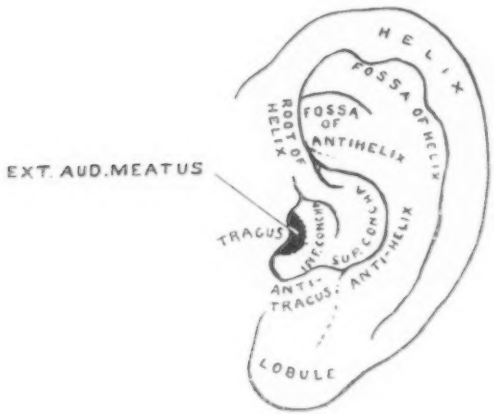

FIG. 2.—The topographical landmarks of the external ear.

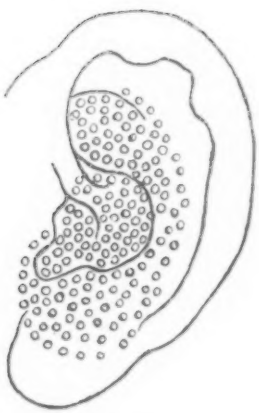

FIG. 3.—The cutaneous zone of the geniculate ganglion on the auricle, outlined by the herpes zoster method.

All of these were associated with a peripheral facial palsy, and in nine of the cases there were auditory disturbances as well. As will be seen by scanning the appended table, the eruptive manifestations in

this series were distributed on one or more of the topographical landmarks of the external ear (see fig. 2), previously indicated as belonging to the geniculate zone, viz., the concha and external meatus, tragus, anti-tragus, incisura intertragica, anti-helix, fossa of the anti-helix, and the superior portion of the lobule, the auditory canal and tympanic membrane (see fig. 3). I would, however, emphasize the fact that in two of the cases, in addition to the typical geniculate eruption in this area, herpetic vesicles were also present in the cleft between the postero-mesial surface of the ear and the adjacent mastoid, in a zone which corresponds to the innervation of the auricular branch of the vagus, and which is a part of the vagal cutaneous zone: but, as the intra-oral distribution of the glosso-pharyngeal vagus (herpes pharyngis et laryngis) in these cases was free from eruption, these retro-auricular

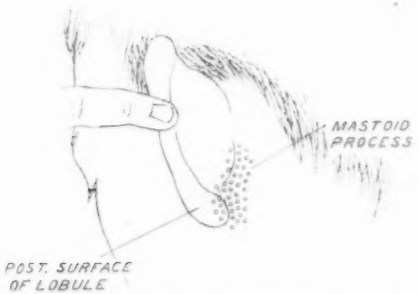

FIG. 4. – The cutaneous strip of the geniculate ganglion on the postero mesial surface of the auricle, outlined by the herpes zoster method.

vesicles may be regarded as probably belonging to the geniculate zone. In other words, the geniculate ganglion, in addition to its zone in the central portion of the auricle, may in certain cases participate in the innervation of a strip of skin covering the postero-mesial surface of the auricle and adjacent mastoid (see figs. 4 and 8). Another point of interest is that in two of the cases, in conjunction with typical herpes in the geniculate area on the external ear, there was associated a unilateral eruption of vesicles within the oral cavity, on the palate just in front of the anterior pillar of the fauces, which confirms the existence of a visceral remnant of geniculate innervation within the buccal cavity, indicated in my previous communications. In this relation the case reported by Frey [12] is especially interesting, in which an eruption of herpes zoster was limited to the tongue in the

chorda tympani distribution, and like the geniculate cases was associated with facial palsy and auditory disturbances. Similar cases have also been described by Siegheim, Bernack, Eichhorst, Remak and Hunt [21], and suggest the possibility of common sensory fibres of geniculate origin passing to the tongue with the chorda tympani nerve.

To state the case briefly: the cutaneous zone of the geniculate corresponds in a general way to the concha auris and also adjacent marginal region, dipping into the auditory canal to the tympanum.

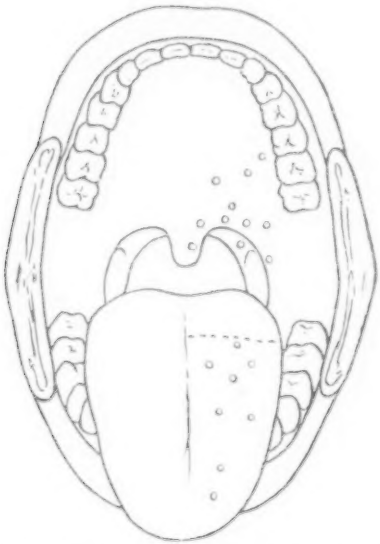

FIG. 5.—The distribution of intra-oral remnants of the geniculate visceral system on the palate and in the chorda distribution on the tongue, as determined by the herpes zoster method.

Its representation in this area is inconstant and variable. It has also an occasional, but quite inconstant, representation on the postero-mesial surface of the auricle and adjacent mastoid, thus sharing with the vagus in the innervation of this region. There is also clinical evidence in support of the occasional occurrence of an intra-oral remnant of geniculate origin on the palate and in the chorda distribution on the tongue (see fig. 5).

In reviewing the extent and distribution of the eruption in my first (eighteen cases) and second series (seventeen cases) of herpes oticus of geniculate origin, certain peculiarities are evident. This zone, so far

as one is able to judge from herpetic manifestations, is small and variable. In certain cases only two or three small groups of vesicles may be present (see fig. 6). In other cases, the entire geniculate zone is fairly well covered, so that it is probable that the zone varies considerably in size and distribution in different individuals (see fig. 7).

FIG. 6.—A case of herpetic inflammation of the geniculate ganglion with herpes otieus and facial palsy. Note the scattered groups of vesicles in the geniculate zoster zone: on the concha, root of helix, anti-helix, and the postero-mesial surface of the auricle: the trigeminal and cervical zones are free from eruption.

This is but natural in an innervation which is regressing phylogenetically, and in this respect is similar to the cutaneous zones of the ninth and tenth ganglia.

It is also significant that the geniculate zone should stand in such close relation morphologically to the small subcutaneous muscles of the auricle, viz., the muscles of the tragus, anti-tragus, and helix, which are themselves regressive and vestigial.

It might be suggested that the area outlined above for the geniculate really belongs to the glosso-pharyngeal-vagus innervation. Against this view may be mentioned the freedom from mucous membrane herpes in the intra-oral distribution of the ninth and tenth nerves and the absence of pharyngeal and laryngeal palsies, while a very important consideration in favour of the geniculate origin is the great frequency of facial palsy, a complication which was present in

FIG. 7.—A case of herpetic inflammation of the geniculate ganglion with herpes oticus and facial palsy. The herpetic eruption is sharply limited to the geniculate zone on the auricle.

practically all of the cases, isolated herpes in the geniculate zone without facial palsy being very rare. It may, however, occur. It has been suggested by Kidd that herpetic involvement of the geniculate ganglion without facial palsy could not occur, and that therefore herpes zoster oticus without paralysis of the seventh nerve could not be of geniculate origin. While it is quite true that facial palsy does accompany herpetic involvement of the geniculate in nearly all cases, that it may occasionally escape involvement must be apparent to those who are familiar with the pathology and symptomatology

of herpes zoster. For example, in rare cases the inflammatory reaction in the affected ganglia may be very slight indeed, acute degenerative changes in the ganglion cells constituting the chief pathological lesion. As for clinical evidence, one has only to recall the isolated involvement of certain groups of cells in the

FIG. 8.—Herpetic inflammation of the geniculate ganglion with herpes oticus and facial palsy. The herpetic vesicles are limited to the posterior cleft between the auricle and mastoid.

Gasserian ganglion, with limitation of the eruption to only one of the branches of the fifth nerve, to realize that the inflammatory reaction within the ganglion may be extremely limited in extent. For these reasons, pathological and clinical, the occurrence of a herpetic inflammatory reaction in the geniculate ganglion with an eruption of herpes zoster and without paralysis of the adjacent motor nerve is not only possible, but probable.

Chapter IV.—The Peripheral Innervation of the Auricular and Intra-oral Zones of the Geniculate Ganglion.

A general outline of the auricular and intra-oral representation of the geniculate ganglion has been given as derived by the herpes zoster method. Particular emphasis was laid upon the variations in distribution as well as the vestigial character of this area. Attention was also directed to the intimate relationship and interlacement with neighbouring sensory zones of the fifth, ninth, tenth and upper cervical ganglia.

The cutaneous zone of the seventh, like the minute intrinsic muscles of the ear, is phylogenetically regressive, and is therefore subject in an unusual degree to anomalies of distribution. It is also significant that this cutaneous sensory remnant should correspond in distribution with those structures which are the outgrowth of the hyoid arch and the spiracle or ear cleft, which is, so to speak, the original domain of the seventh nerve. The external ear is developed from a series of small rounded prominences or auditory tubercles which originate in the mandibular and hyoid arches. The mandibular arch lies within the trigeminal area, while the hyoid arch is included within the area innervated by the auricular branches of the seventh, ninth, tenth and cervical nerves. As the seventh nerve is, properly speaking, the nerve of the first branchial cleft, its remnant of innervation in man represents the persistence of what in the lower forms was a larger and more important area.

The exact course pursued by the cutaneous fibres passing from the external ear to the geniculate ganglion is as yet unknown, and further experimental, anatomical and embryological investigations will be required in order to settle definitely this intricate question. I will, however, present my own views upon this subject, based on such facts as I have been able to gather, and while only tentative and requiring additional proof, they may be of assistance to those who approach the problem from another point of view.

On phylogenetic grounds it would seem probable that cutaneous fibres pass out with the main trunk of the facial nerve and emerge with the motor fibres at the stylo-mastoid foramen. That the facial at this point contains sensory fibres has been demonstrated experimentally by Van Gehuchten [44] and by Weigner [46].

One of several routes may be taken by the cutaneous fibres of the seventh after leaving the main trunks. (See fig. 1.)

(*a*) Through the communications which take place between the facial and the auricular branch of the vagus in the lower portion of the Fallopian canal.

(*b*) With the posterior auricular branch of the seventh nerve.

(*c*) Through communications between the main trunk of the seventh nerve and the auriculo-temporal branch of the fifth; and with the motor filaments destined for the innervation of the minute intrinsic muscles of the external ear.

(*a*) In "Quain's Anatomy" [35] the distribution of the auricular branch of the vagus is described as follows: "The auricular branch (nerve of Arnold) arises from the ganglion of the root (ganglion jugulare); it receives the filaments from the petrosal ganglion of the ninth: it then enters and traverses the substance of the temporal bone, crosses the aqueduct of Fallopius about 4 mm. from the lower end, forming a communication with the facial, and finally emerges between the external auditory meatus and the mastoid process. It divides into two parts, one of which joins the posterior auricular innervation of the seventh, while the other is distributed to the skin on the back of the pinna and the lower back parts of the auditory canal.[1] A similar distribution and connexion with the facial nerve is accorded the auricular branch of the vagus in "Testut's Anatomy." (See fig. 9.)

In the description of the collateral branches of the seventh nerve, Quain also states that "a communication with the auricular branch of the vagus is generally present in the form of a twig leaving the facial close above the stylo-mastoid foramen."

(*b*) The posterior auricular branch of the seventh nerve is given off as it emerges from the stylo-mastoid foramen. It then passes backwards and upwards to the anterior border of the mastoid process, and there divides into two branches — an ascending and a horizontal. The ascending or superior branch courses upward between the mastoid process and the auricle and terminates in the superior and posterior auricular muscles. The horizontal or posterior branch passes backwards and forwards and is distributed to the occipital muscle. Valentin [43] has described cutaneous filaments to the auricle from the ascending and horizontal branches of the posterior auricular branch of the facial nerve.

[1] Quain, in discussing the variations and anomalies in this region, states: "In rare instances, absence of the auricular branch of the vagus has been observed, or of the communication with the facial nerve. The auricular branch occasionally passes entirely into the facial trunk, and in that case its fibres are probably conveyed to the external ear through the posterior auricular nerve."

(c) Anastomotic filaments uniting the temporo-facial branch of the seventh with the auriculo-temporal branch of the fifth. As the facial nerve splits up into its terminal divisions in front of the ear, small anastomotic filaments pass to the neighbouring auriculo-temporal branch of the fifth. As the auricular branches of this nerve pass

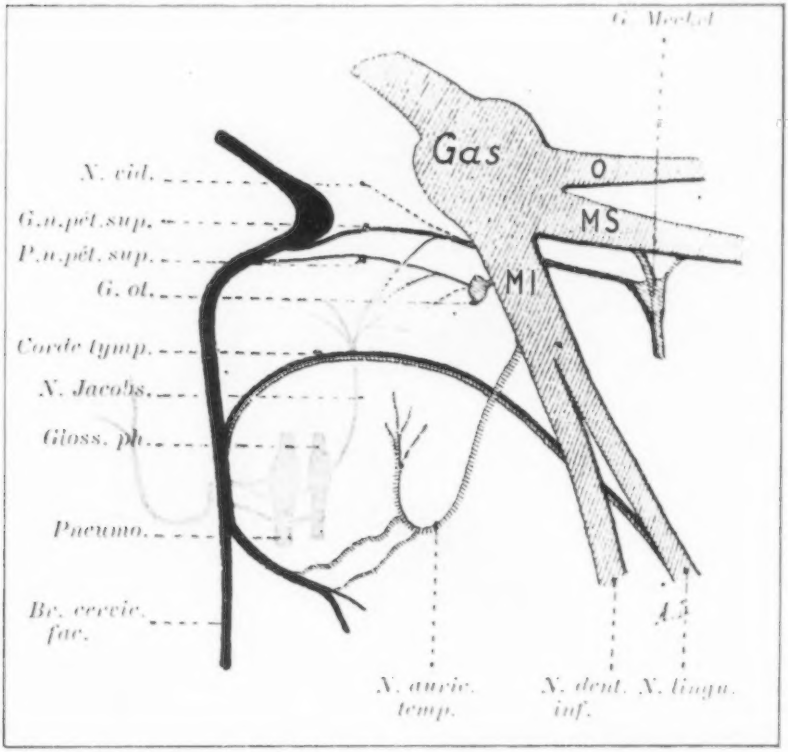

FIG. 9.—From Testut, "Anatomie humaine," 1905, vol. iii. A schematic representation, showing the anastomoses of the facial with the trigeminal, and the auricular branches of the glosso-pharyngeal and pneumogastric nerves.

to the skin of the tragus, superior portion of the helix, and the anterior portion of the auditory canal and tympanum, it would be possible for sensory fibres of geniculate origin to reach the external surface of the pinna by this route. Some sensory fibres may also course with the motor filaments of facial origin which supply the minute subcutaneous muscles of the auricle.

That cutaneous fibres of geniculate origin should reach the external ear by way of the petrosal nerves and their communications with the trigeminus is, on phylogenetic grounds, very unlikely. It is especially the communications between the facial and the auricular branch of the vagus, the posterior auricular nerve, and the anastomoses with the auriculo-temporal branch of the fifth, to which I would direct attention as the probable pathways of these sensory filaments.

In the classical text-book descriptions of the external auditory canal and the outer surface of the tympanic membrane, the nerve supply is divided between the auricular branches of the trigeminus and the auricular branch of the vagus, the trigeminal area occupying approximately the anterior half of the canal and tympanic membrane, while the vagus is accorded the posterior strip. In my descriptions of the extent and distribution of the zoster zone of the geniculate, it was pointed out that the facial in some cases retains a remnant of innervation within the auditory canal and on the typanum. While the zoster evidence in favour of this view is by no means conclusive, it is nevertheless suggestive and should be considered in anatomical investigations in this subject (Wilson [47]). It would be strange indeed if the seventh nerve had no sensory remnant in a domain which was formerly its own (first branchial cleft), while the auricular filaments from the ninth and tenth were still represented in no inconsiderable degree. Indeed, were it not for the traditional anatomical point of view, founded essentially on the dissection method and the motor symptomatology of the seventh nerve, one's first thought would have been of the geniculate ganglion in connexion with the innervation of the structures developing from the hyoid arch.

Intra-oral representation of the geniculate ganglion.—That in man sensory remnants should still persist in what was formerly an important visceral innervation of the seventh within the buccal cavity is not surprising. The herpes zoster evidence indicating the presence of such rudiments consists in the occasional occurrence of an eruption limited to the anterior two-thirds of the tongue in the distribution of the chorda tympani, and on the palate in the region of the anterior pillar of the fauces. Cases of this kind were analysed in my earlier contributions and more recently similar observations have been reported by Palmer [34] and Closier [6]. In Palmer's case (see table) the eruption was distributed on the concha, the posterior wall of the canal, the anti-helix and fossa of the anti-helix, i.e., in the geniculate auricular zone; but in addition there was also an eruption

on the anterior pillar of the fauces and the adjacent region of the soft palate. The herpes was accompanied by facial palsy and acoustic nerve symptoms. An almost identical case is reported by Closier, the herpetic eruption on the ear appearing on the concha, tragus, external meatus, and the posterior wall of the canal, but in addition herpetic vesicles were also present on the soft palate. This case, like the preceding, was associated with facial palsy and auditory symptoms. It is, therefore, probable that the geniculate retains in some cases a remnant of its former sensory function within the buccal cavity, in the area of distribution of the chorda tympani and in the region of the soft palate. It will be recalled that many years ago Bernhardt pointed out the rare occurrence of hypæsthesia in the chorda distribution in cases of facial palsy, an observation which was subsequently confirmed by Harvey Cushing [7] in his studies of the trigeminal zone; these observations confirming by another method and in a different group of cases the conclusions which I had reached in regard to the intra-oral visceral remnant of the seventh nerve.

In explanation of the palatal herpes and its possible relation to the geniculate ganglion there would seem to be some anatomical basis in the fact that the lingual branch of the seventh, which innervates the stylo-glossus and palato-glossus muscles at the base of the tongue, also sends mucous filaments to the anterior pillar of the fauces and adjacent region. And, while it is true that this lingual branch has an anastomosis with the glosso-pharyngeal, there is no reason to assume that these mucous filaments are not a part of the sensory facial system and originating in cells of the geniculate ganglion. It is also possible that sensory fibres of geniculate origin pass to the oral cavity by way of the petrosal nerves and their connexions with the trigeminus.

The geniculate ganglion, therefore, in addition to its taste function, has a variable and vestigial intra-oral sensory representation, as have the glosso-pharyngeal and vagus nerves; and all three of these branchial nerves, the seventh, ninth and tenth, are represented both within the mouth cavity and on the external ear.

Chapter V.—Comparative Anatomy and its Bearing on the Cutaneous Zone of the Facial Nerve.

Of great importance in the final elucidation of this problem of the cutaneous innervation of the facial nerve will be the work of the comparative anatomist. A large literature already exists on the subject of the comparative morphology of the cranial nerves, and the separation

and description of their various nerve components by the comparative method. One of the fruits of such investigations has been the demonstration in man of the *nervus terminalis*, which had been traced successively through many of the lower forms. (Johnston [24]). Even as low in the verbetrate scale as fishes, the *nervus terminalis* is vestigial. I mention this instance of the curious persistence of a rudimentary cranial nerve in man as bearing upon the subject under discussion. It is my belief that the cutaneous component of the seventh nerve may also be traced upward through the vertebrate scale by investigation in the field of comparative neurology. At the present time the tendency has been to deny the existence of a cutaneous component of the seventh nerve in forms above the *cylostomes*, notwithstanding the fact that cutaneous fibres are found in the seventh nerve of selachians, teleosts and amphibians, but are usually supposed to originate from the neighbouring sensory systems of the fifth and tenth nerves. One reason for this point of view, I believe, is the traditional anatomical conception that the external ear is innervated by the trigeminus anteriorly, the cervical nerves posteriorly, with a small intervening field of the glosso-pharyngeal and vagus, so that it was very easy and natural to regard the few cutaneous fibres in the seventh nerve as accessions from the trigeminal or vagal systems. Already, however, there are indications of a change from this point of view.

In a recent study of "The Cranial Nerves of Siren Lacertina," H. W. Norris [33], in the chapter entitled "The General Cutaneous Component of the Facial Nerve," writes as follows: "The presence of general cutaneous fibres in the seventh nerve of amphibians has been commonly recognized, but no distinct route of the facialis entering the spinal fifth tract has been found. It is generally assumed by students of nerve components that any and all such fibres found their way into the seventh nerve from the tenth nerve through the ramus communicans cum facialis. To the writer's knowledge no one has found general cutaneous fibres in the root of the facial nerve. Any suggestion therefore of the occurrence of such fibres challenges contradiction and calls for a most critical examination of the so-called evidence." Norris then proceeds to describe such a cutaneous component in *siren*, and his conclusions are as follows: "The apparent occurrence of the general cutaneous constituent in the facial nerve roots also suggests that it is possibly more generally present in other *urodela*, but because of its minuteness and close association with other components has hitherto escaped unnoticed. Its presence is best

explained on the ground of a persistence of a function and at one time more fully developed characteristic of the facial nerve."

Judson Herrick [17], in a study of the medulla oblongata of larval *amblystoma*, in a chapter describing "General Cutaneous Roots of the Fifth, Seventh and Ninth Nerves," says: "In a number of longitudinal sections by the methods of Cajal and Golgi I have seen evidence of a few fibres from the seventh root complex entering the spinal fifth root. In one Golgi impregnation such a fibre is clearly defined for a long distance. This fibre bifurcates into ascending and descending branches, both of which enter the spinal fifth root. These fibres probably represent the general cutaneous component of the facialis described by Norris in *siren*, though I have not succeeded in following them peripherally, or recognizing their ganglion, and my observations are so few as to require further control."

Kingsbury [26], as far back as 1897, demonstrated the central connexions of the cutaneous component of the seventh nerve in ganoid fishes, and it is not unlikely that in the near future more anatomical evidence of this nature will be forthcoming, and will confirm the results obtained in man by purely clinical methods of study.

Chapter VI.—On Disturbances of Sensibility in the Sensory Field of the Facial Nerve.

Conchal hypæsthesia.—The disturbances of sensibility in the geniculate zone after lesions of the facial nerves are slight and comparatively insignificant, because of the small area involved, its rudimentary character, and the overlap from the neighbouring systems of the fifth, ninth and tenth, and upper cervical ganglia. It is also probable from the herpetic manifestations that anomalies and variations in size of the geniculate area are not uncommon. In my earlier contributions to the sensory system of the seventh nerve, attention was called to an area of hypæsthesia in the concha of the ear which was sometimes demonstrable in recent cases of facial paralysis. As was stated at that time, "this disturbance of the sensibility in the conchal region never reached the degree of anæsthesia. The tactile sense was merely diminished or obtunded in this area, and care was required to demonstrate its presence." Since that time I have repeatedly demonstrated the existence of this small area of conchal hypæsthesia in recent cases of facial neuritis, and occasionally after herpetic inflammations of the geniculate ganglion. This area of hypæsthesia, however, is not

permanent and disappears early, and cannot be sharply outlined, as in trigeminal anæsthesia; nor, indeed, was this to be anticipated, considering the small size of the area involved, and its interlacement with adjoining sensory zones.

Kidd in his remarks on the cutaneous zone of the seventh nerve makes this statement: "As a matter of fact, if there were any facialis cutaneous fibres in man, we should find in every case of Bell's palsy as complete a cutaneous anæsthesia of the alleged auricular zone as any clinicians have found on the trigeminal cutaneous area after a complete Gasserianectomy." But this surely is not necessary, nor even probable, and had Kidd been familiar with my later work on the zoster zones of the geniculate, glosso-pharyngeal and vagal ganglia [21], which is not mentioned in his article, this statement might have been considerably modified.

Of special interest from its bearing on the sensory disturbances of the external ear is the case of geniculate otalgia reported by Clark and Taylor [2], in which a cure was effected by section of the nerve of Wrisberg. Kidd, who has utilized this case in his discussion of the cutaneous zone of the seventh nerve, approaches this part of his criticism as follows: "And yet, amidst all the well-deserved rejoicings that followed the recital of this case on this New York joy-day, in which Ramsay Hunt took part, actually no speaker asked the question as to what was the post-operative state of cutaneous sensibility on the auricle, and there is even no mention as to whether it was ever tested!" Here again, Kidd indulges in a rather rash statement which is based on an incomplete knowledge of the literature of the subject which he is discussing. The occasion to which he refers was a *later* presentation of the patient to the New York Neurological Society, November 9, 1909 (Clark and Taylor [1]), and which was some months after the original presentation to the American Neurological Association in May, 1909. The case was reported in full in the *Journal of the American Medical Association*, December 25, 1909 (Clark and Taylor [2]), in which the following description of the sensory examination was given: "All sensory examination of the face and external ear proved negative three days after operation. The former area of pain seemed to the patient to be a little less sensitive in the test." So that in this case, while there was no well-defined sensory disturbance, there was a lessened sensibility in the auricular area which had been the seat of pain and which harmonizes with the slight disturbances of sensibility demonstrable after other lesions (seventh palsy and ganglion inflam-

mations) and is in accord with the rudimentary and variable character of the geniculate zone.

Hypæsthesia on the postero-mesial surface of the auricle and adjacent mastoid.—It is occasionally possible to demonstrate in conjunction with conchal hypæsthesia a diminution of sensibility on the postero-mesial surface of the auricle and adjacent mastoid region in cases of recent facial palsy. This area corresponds to the auricular distribution of the vagus and that of the ascending filament of the posterior auricular branch of the seventh nerve, which, according to Valentin, carries cutaneous filaments to this region. A hypæsthesia in this region may be caused either by involvement of the sensory filaments of the seventh nerve, or of the auricular branch of the vagus as it crosses the facial at the lower end of the Fallopian aqueduct.

As has already been stated, this area is sometimes the seat of herpes in cases of geniculate inflammation, and it is probable that the seventh and tenth ganglia both have rudimentary representations in this post-auricular strip (see fig. 4).

Sensory disturbances of the face and neck in cases of geniculate ganglion inflammation.—In the interpretation of sensory disturbances in the geniculate zone in cases of herpetic inflammations, it should not be forgotten that in some cases there is an associated hypæsthesia and hypalgesia in the trigeminal distribution and occasionally in the cervical zones as well. This I have regarded as evidence of a mild inflammatory reaction in neighbouring ganglia, and has been fully discussed in an earlier paper in the chapter, "Multiple Involvement of Ganglia in Zona" [22]. Such hypæsthesias are often accompanied by darting pains, loss of the corneal reflex, and groups of aberrant zoster vesicles, all of which are additional indications of ganglionic involvement. It is also well to recall that in cases of facial palsy there is not infrequently a diminution in the sensibility of the face on the paralysed side which has been variously interpreted: as of hysterical or psychogenic origin, as a functional derangement of the ganglion of the fifth nerve induced by motor insufficiency of the paralysed face; as the result of hypotonia of the cutaneous muscles with consecutive feebleness of the dermal papillæ; as a neuritis of peripheral sensory fibres passing from the trigeminus to the facial muscles. It should, however, be emphasized that whatever the underlying cause, psychogenic, physiological, or neuritic, of this mild hypæsthetic disturbance, it should not be identified in any way with the geniculate area.

Intra-oral sensory disturbances of geniculate origin have as yet not

been demonstrated, except for the rare occurrence of hypæsthesia in the chorda distribution of the tongue, as was shown by Bernhardt in cases of recent facial palsy. This observation has been confirmed by many since Bernhardt first drew attention to it, and all agree that it is slight and very inconstant.

It is probable that routine examinations will show a similar inconstant hypæsthesia on the palate near the faucial margin, but thus far I have not encountered it (*see* fig. 5).

On two occasions the passage of a sound into the Eustachian tube was less sensitive on the side of an inflammation of the geniculate ganglion with facial paralysis. This occurrence I was inclined to explain by the participation of the sensory system of the geniculate in the innervation of the tympanic cavity and its prolongations, the Eustachian tube and mastoid cells.

Chapter VII.—Recapitulation and Concluding Remarks.

The facial nerve is, therefore, to be regarded as a mixed cranial nerve, homologous with the trigeminal, glosso-pharyngeal, and vagal systems. In common with these, the facial possesses a definite group of symptoms and syndromes directly dependent upon involvement of its sensory system.

In the lower forms of life the sensory facial equals, and may even exceed in importance, the motor functions. In man, however, the conditions are reversed—the motor function is predominant, and its sensory system has retreated before the advance of the trigeminus in front and the cervical areas posteriorly.

In comparative anatomy the facial is the nerve of the first branchial or ear cleft, and evidences of this ancient phylogenetic relationship is still present in man, in the cutaneous remnant which persists on the external ear as well as its relations to the other structures of the auditory mechanism which take their origin from the region of the spiracle or first visceral cleft.

That portion of the facial sensory system which still persists in man is concerned in the innervation of the internal ear, the middle ear, and its prolongations into the Eustachian tube and mastoid cells, and the skin on certain portions of the external ear. There is also evidence that a vestigial remnant occasionally participates in the innervation of a strip on the postero-mesial surface of the auricle and within the buccal cavity in the chorda tympani distribution, and on the palate near the anterior pillar of the fauces.

The sensory system of the geniculate ganglion has the following symptomatology:—

(*a*) Pain and sensory disturbances in facial neuritis.

(*b*) Neuralgic affections; primary tic douloureux, reflex otalgia, herpetic and tabetic otalgia.

(*c*) A sensory pathway for the direct transmission of irritative impulses to the facial nucleus, thus favouring the production of facial twitchings and spasms.

(*d*) Herpetic inflammations of the geniculate ganglion, a *syndrome* characterized by an eruption of herpes zoster in the geniculate zone, usually associated with facial palsy and often with auditory symptoms.

The cutaneous zone of the geniculate ganglion is represented on one or more of the following landmarks of the external ear (see figs. 2 and 3), viz., the concha, external meatus, tragus, anti-tragus, incisura intertragica, anti-helix, fossa of the anti-helix, superior portion of the external surface of the lobule, within the auditory canal and on the tympanic membrane. There is also reason to believe that the geniculate may in certain cases participate with the ninth and tenth ganglia in the innervation of a cutaneous strip on the postero-mesial surface of the auricle and adjacent mastoid region (see fig. 4).

In this outline of the cutaneous zone it is not to be assumed that this entire region is exclusively innervated by the geniculate ganglion, but merely that the geniculate zone which is variable in size and distribution falls within this area, and that other sensory systems, e.g., the fifth, ninth, tenth, and cervical nerves, also participate. Furthermore, as the cutaneous zones of the seventh, ninth, and tenth are rudimentary, there must be considerable differences in size and relationship in individual cases, and these variations in size and distribution, as well as the overlap of neighbouring systems, must be considered in the interpretation of their respective zones.

The same is true of the intra-oral remnant of the seventh nerve in the chorda distribution and in the region of the palate near the anterior pillar of the fauces, both of which are inconstant and variable (fig. 5).

The sensory disturbances following lesions of the facial nerve in in the Fallopian canal have the following characteristics: The pain is situated in the depths of the ear and mastoid region and in the central portions of the external ear, sometimes extending in the direction of the throat and tonsil. When severe, irradiation may also occur in the trigeminal, occipital and cervical areas. In some recent cases of facial palsy and occasionally after geniculate inflammations, a hypæsthesia in

the region of the concha is demonstrable. It is slight and cannot be definitely delimited. Rarely in seventh palsies a postero-mesial strip on the auricle is also hypæsthetic. The inconstancy and moderate degree of the sensory disturbances when present are dependent upon the rudimentary nature of the sensory field of the geniculate, its variations in size, and its interlacement with the auricular branches of neighbouring distributions. For these reasons the herpes zoster method furnishes the best clinical criteria of its probable extent and variation.

It has been suggested that an eruption occurring within what I have described as the geniculate zoster zone may be of trigeminal, cervical, or glosso-pharyngeal vagal origin. That the occurrence of isolated herpes zoster oticus in this zone should be of either trigeminal or cervical origin is readily excluded by the small size and the peculiar location of the eruptive manifestations. Some of the cases are certainly of glosso-pharyngeal and vagal origin, and elsewhere the attempt has been made to isolate and separate their zones from those of geniculate origin. That the other cases are certainly of geniculate origin is shown by the almost constant association of facial palsy due to the proximity of the motor fibres of the seventh to the affected ganglion. Were these cases of glosso-pharyngeal or vagal origin, facial palsy should not occur so constantly. One would anticipate rather pharyngeal and laryngeal palsies or symptoms of vagal irritation (McKenzie [48]). Indeed it would be in just such lesions of the vagal ganglion that one would expect symptoms of pneumogastric irritation, e.g., coughing, singultus, bradycardia, nausea, and vomiting, and yet these distinctively pneumogastric symptoms were absent in the thirty-five cases of pure herpes oticus which I analysed in differentiating the geniculate zone.

As my results are based very largely on the analysis and study of cases by the "herpes zoster method," they cannot be accepted as the final solution of this difficult problem. Many more observations will be required in order to determine more accurately the extent of these auricular and intra-oral zones and their relationship, as well as further experimental and morphological studies.

It is, however, a beginning and may serve as a guide in future investigations.

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BILATERAL LESION OF THE OCCIPITAL LOBES WITH RETENTION OF MACULAR AS DISTINCT FROM PANORAMIC VISION.

CLINICAL RECORD

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Clinical Record.

The patient, a man, aged 45, was sent to me (B. B.) by Dr. (now Sir) George A. Berry in February, 1886, complaining of loss of sight. Dr. Berry reported that there was nothing wrong with his fundus. The loss of sight, which consisted of complete loss of peripheral vision with very marked reduction of macular vision, had come on suddenly with an attack of what appeared to be uræmic convulsions in June, 1884.

I diagnosed the condition as probably due to a bilateral lesion of the occipital lobes in the region of the half-vision centre. The case did not appear to be one of the rare cases of double homonymous hemianopsia, in which a lesion of one occipital lobe (say the right) produces a homonymous hemianopsia on the opposite (say the left) side, and a second and subsequent lesion in the other (say the left) occipital lobe produces a homonymous hemianopsia on the opposite (say the right) side, but a *simultaneous* bilateral lesion in the right and left occipital lobes—an infinitely rare, if not a unique, condition.

I recorded the case in my "Atlas of Clinical Medicine" (vol. ii, p. 49), published in the year 1893 (seventeen years before the patient's death), and there stated:—

"This is a most interesting case. In the absence of a post-mortem examination it is, of course, difficult to say with any degree of certainty what is the position and pathological character of the lesion. But the condition of the visual defect—complete abolition of peripheral vision, with, comparatively speaking, good central vision both for white and for colours—and the absence of any changes in the fundus oculi are, I think, strongly suggestive of a bilateral lesion of the half-vision centre in the back part of each occipital lobe."

I watched the patient carefully until his death on October 21, 1910, a period of twenty-four and a half years, making it worth his while to see me every six months, in order that I might keep in touch with his condition. During the whole period (twenty-four and a half years) that the patient was under my observation the condition remained, practically speaking, unchanged, and there were no further cerebral developments; central (macular) vision improved slightly, peripheral vision remained completely obliterated. Throughout the whole course of the case there was no motor paralysis, no loss of sensation, and no aphasia, either sensory or motor.

In April, 1905 (twenty-one years after the onset of the lesion, and five years before the patient's death) Dr. A. H. H. Sinclair kindly examined the patient and gave me an exhaustive report on the condition of vision and of the fundi, and accurate charts of the fields of vision (figs. 3 and 4).

The patient died of acute croupous pneumonia on October 21, 1910, aged 71. Through the kindness of Drs. George Donald and George M. Johnston, of Leith, I was able to make a post-mortem examination.

A large lesion was found in each occipital lobe.

The case seemed to me to be of so much scientific interest that I asked Dr. J. Shaw Bolton to undertake the microscopical examination. This he very kindly consented to do.

It is worthy of note, considering the great extent of the lesion in both occipital lobes and the extensive severing of the connexions between the visuo-sensory centre and the other parts of the brain, that there was no mind-blindness.

The case is, as Dr. Shaw Bolton states, unique and of the highest scientific value. It was carefully studied during life and watched for a period of twenty-four and a half years by myself; the ocular and visual

condition was carefully noted by two leading authorities on ophthalmic medicine—at the commencement of the case by Dr. (now Sir) George A. Berry and at the end of the case by Dr. A. H. H. Sinclair; and the histological investigation, involving as it has done an enormous amount of time and labour, was carried through by the leading authority on the histology of the visual area and occipital lobe, Dr. J. Shaw Bolton, assisted by Dr. William Robinson.

The details of the case are as follows:—

Acute general dropsy (probably due to acute nephritis); epileptiform convulsions; permanent loss of peripheral vision, the fields being enormously contracted: central vision both for white and for colours fairly good; fundi oculi normal; no mind-blindness and no word-blindness. The diagnosis, made twenty-four years before death, was "probably a bilateral lesion in the occipital lobes"; death at the age of 71, twenty-six and a half years after the onset, from acute croupous pneumonia. During these twenty-six and a half years there were no fresh head symptoms, and practically no change in vision, no motor paralysis, no loss of sensation, no aphasia, sensory or motor. On post-mortem examination a large lesion was found in each occipital lobe.

M. M'G., a labourer, aged 45, was sent to me on February 24, 1886, complaining of loss of sight.

Previous history.—The patient stated that he had enjoyed good health until June, 1884, when he had a severe illness, which seemed to have been acute nephritis with uræmia. The attack began with bronchitis and "stoppage of the water" (he does not know if the water was red or bloody); his face, body, and limbs swelled, and he lost his appetite. During the course of the attack he suddenly lost his eyesight, and almost immediately afterwards had a series of epileptic fits. He was unconscious for fourteen days; it was three weeks before he could recognize voices. When he regained consciousness he was quite blind; since then his eyesight (central vision) has been gradually restored in some degree.

Since he recovered from the acute stage of the attack his general health has been good; he now feels well, if it were not for the loss of sight; he is, however, "at times heavy and inclined to sleep." For some time after he recovered from the acute attack he occasionally suffered from headache, but the pain was not frequent and not severe; for the past year there have been no headaches. He has not suffered from giddiness or vomiting. His face has never swelled since he recovered from the acute attack. He has not had syphilis.

Condition on February 24, 1886.—The urine was absolutely normal, and the heart and other organs quite healthy; the patient was muscular and well nourished. There was no motor paralysis, no loss of sensation, and no aphasia, either sensory or motor.

The knee-jerks were normal; there had been no lightning pain; the pupils were of medium size, they contracted actively both to light and accommodation—in short, there was no suspicion of tabes.

Central vision for white was very defective (V. $\frac{3}{18}$). The peripheral portions of the field were so greatly constricted that peripheral vision was *nil*.

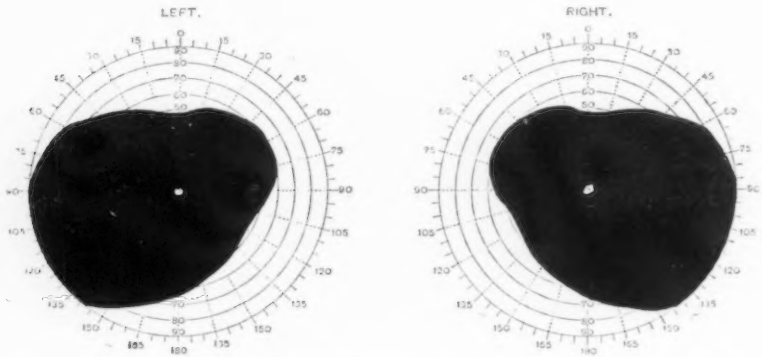

FIG. 1.—Perimeter chart of the fields of vision in the case of M. M'G., described in the text, taken on January 12, 1887. (Reduced from 9½ in. to 4½ in.)

There were no ophthalmic changes—a fact confirmed by Dr. Berry.

Subsequent progress of the case (*January* 12, 1887).—The patient's general health is good. His sight is better; he says that he can see little objects, such as a pin on the floor, but that he fails to see larger objects, and often runs up against things, tumbles over his children, &c. Central vision has improved—right eye $\frac{5}{18}$ imperfectly; left eye $\frac{3}{18}$ imperfectly; central colour vision good. The condition of the fields at this date (January 12, 1887) is shown in fig. 1.

March 19, 1891.—His general health is good. The urine continues quite normal; there have been no head symptoms—in fact, no nervous symptoms at all, except the affection of sight. He stated that he sees objects distinctly when they are placed directly in front of him; and that he can see small objects, such as a pin or a small piece of white paper the size of a pea on the floor. I satisfied myself that this state-

ment was correct. In order to see a small object on the floor in front of him he moves his head and eyes about (ranges over the area where he knows the object is) until he gets it in the line of central vision.

Central vision is fairly good—much better than it was four years ago; at 18 ft. he correctly named with the right eye one of the test letters which ought to be read at 18 ft., and three out of five of the letters which ought to be read at 24 ft. (Right eye, V. $= \frac{18}{24}$ imperfectly.)

With the left eye he could not recognize any of the letters which ought to be read at 18 ft., but at this distance he correctly named three of the five letters which ought to be read at 24 ft. (Left eye V. $= \frac{18}{24}$ imperfectly.)

Vision was not improved by glasses.

Central vision for colours was perfect in each eye.

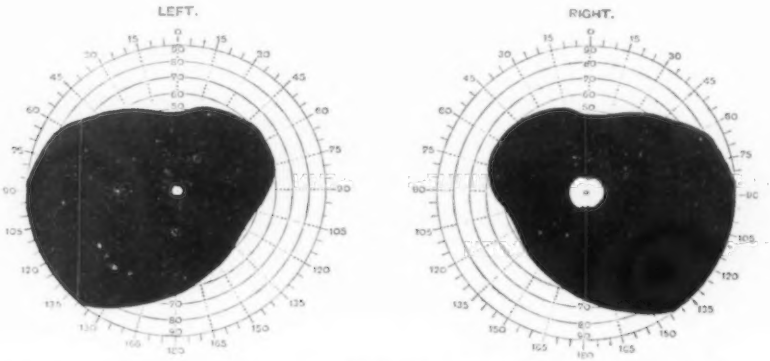

FIG. 2.—Perimeter chart of the fields of vision in the case of M. M'G., described in the text, taken on March 19, 1891. (Reduced from 9½ in. to 4¼ in.)

He says he cannot see anything at the sides, above or below. The fields of vision were again mapped out with the greatest care; the area of central vision was a little, but only very little, larger than on January 12, 1887; the improvement was most marked in the right eye (see fig. 2).

The pupils contracted actively both to light and on convergence.

There were no abnormal ophthalmoscopic appearances.

The day on which these observations were made was particularly clear and bright.

His mental condition is not very bright, and he says his memory is

not good, but there is no mind-blindness and no word-blindness. He walks about the streets without assistance, coming up every six months by himself to the Royal Infirmary to see me. He can read large type, such as the prices of objects in shop windows.

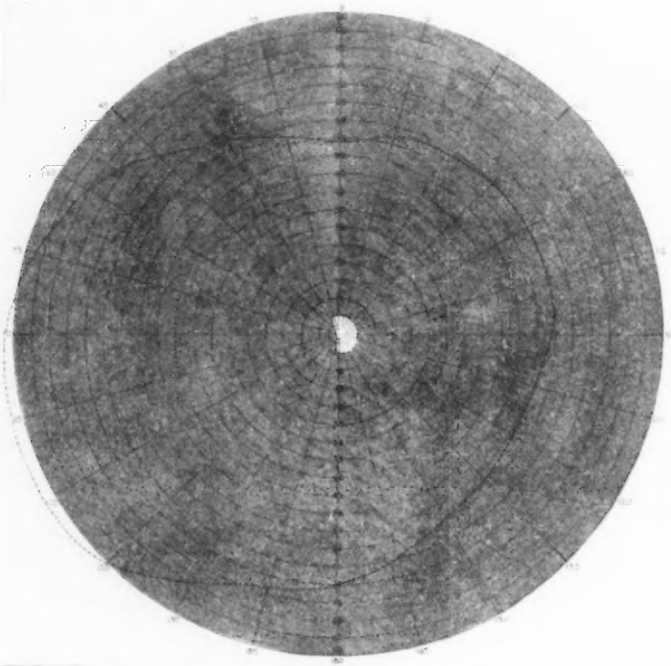

FIG. 3.—Perimeter chart of the *left* field of vision in the case of M. M'G., taken by Dr. Sinclair on April 10, 1905. (Reduced from 7½ in. to 4 in.)

The dark interrupted line encloses the area over which the field of vision normally extends. The part of the dark shading within the dotted line shows the extent to which blindness had encroached on the field, and the area at the centre, which is very lightly shaded, indicates the remaining field. The remaining field responded to a white test object 10 mm. in diameter and seen at 250 mm. distance (as taken with the perimeter). The small white areas within the remaining field on the right side of the fixation point responded to a smaller stimulus, i.e., a white test object 10 mm. in diameter and seen at 2,000 mm. distance on Bjerrum's screen.

On May 19, 1891, the patient was admitted to the Edinburgh Royal Infirmary suffering from scurvy, under Professor Wyllie, who has kindly allowed me to see his notes. The attack had commenced twelve days previously, and had evidently produced marked (temporary)

deterioration of vision and of the mental condition. The patient was stupid, his memory was poor, the vision very defective. He was discharged cured of the scorbutus on June 18, 1891.

January 8, 1893.—There is no change in the general condition vision is not so good as it was on March 9, 1891.

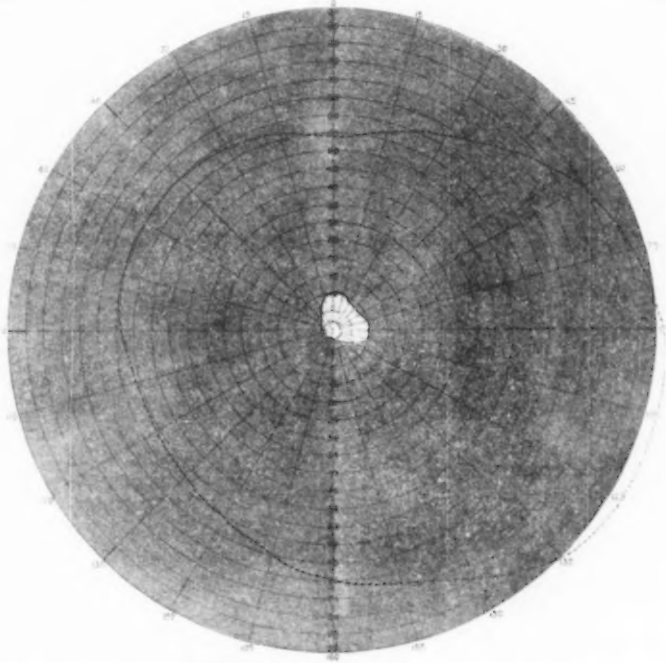

FIG. 4.—Perimeter chart of the *right* field of vision in the case of M. M'G., taken by Dr. Sinclair on April 10, 1905. (Reduced from 7½ in. to 4 in.)

The dark interrupted line encloses the area over which the field of vision normally extends. The part of the dark shading within the dotted line shows the extent to which blindness had encroached on the field, and the area at the centre, which is very lightly shaded, indicates the remaining field. The remaining field responded to a white test object 10 mm. in diameter and seen at 250 mm. distance (as taken with the perimeter). The small white areas within the remaining field on the right side of the fixation point responded to a smaller stimulus, i.e., a white test object 10 mm. in diameter and seen at 2,000 mm. distance on Bjerrum's screen.

April 26, 1894.—Does not feel so well; thinks his sight is not so good; is occasionally giddy; a strange feeling—"sort of weakness"—comes over him at times. His memory, he says, is impaired, but his visual memory (for places, objects, &c.) is not specially interfered with.

He walked up alone from Leith to the Edinburgh Royal Infirmary to-day.

April 8, 1905.—*In statu quo.* The patient continues to comes up to the Infirmary to see me every six months, as he has done regularly during the past twenty years. General health good.

Dr. Sinclair kindly made an exhaustive examination, and sent me the following report on the ocular condition and vision:—

(*Dr. A. H. H. Sinclair's Report, April* 10, 1905.)

There is some slight chronic conjunctivitis present in both eyes—right eye, V. $\frac{6}{36}$ uncertainly; left eye, V. $\frac{6}{36}$ and $\frac{6}{24}$ partly. Central fixation is present, but is not perfect.

The field of vision is very greatly contracted, and more so on the left side in both eyes (see figs. 3 and 4). The fixation point is probably encroached upon on the left side in both eyes. The slightly larger extent of the right field corresponds to the patient's statement that he can see "a little wider with the right."

The pupils are small (3 mm.) react to light, and are equal.

The eyes move normally. Convergence and other movements are uncertainly performed, because of the failure in indirect vision.

The *ophthalmoscopic examination* shows both discs to have a somewhat leaden-grey atrophic appearance, with extensive shallow cupping, probably more than the original physiological cupping.

There is a well-developed scleral ring round each disc—quite like the high-water mark seen round the disc in glaucoma; though not in this case connected with glaucoma, it is an evidence of the atrophic process which apparently has been going on for a long time.

The general fundus shows in each case some tessellation of the choroid. In the left eye there are some slight changes in the choroid between the disc and the macula.

The retinal arteries have a hard "silver-wire" look, but otherwise the retinal vessels are normal.

On June 15, 1905, the patient was admitted to the Edinburgh Royal Infirmary, under my care, suffering from scurvy of a fortnight's duration. He was discharged cured of the scorbutus on July 7, 1905.

June 2, 1910.—No change since the last note; general health good; walked up from Leith alone as usual to see me to-day; his memory, he says, is no worse; can read large type; can write, but does not write letters—his sight is so bad that the lines run into one another.

After the patient's death his daughter, with whom he lived, told me that she remembered his having one convulsion of recent years. He could, she said, read large type, and could tell the prices of objects in the shops. He only once, so far as she remembered, wrote a letter; it was very bad, for he wrote one line on the top of another. He never complained of his head; his memory was good, and his mind was all right. He was able to find his way about quite well.

The patient died, at the age of 71, from acute croupous pneumonia on October 21, 1910. Through the kindness of Drs. Donald and Johnston I was informed of the patient's death, and was able to get permission for a post-mortem examination.

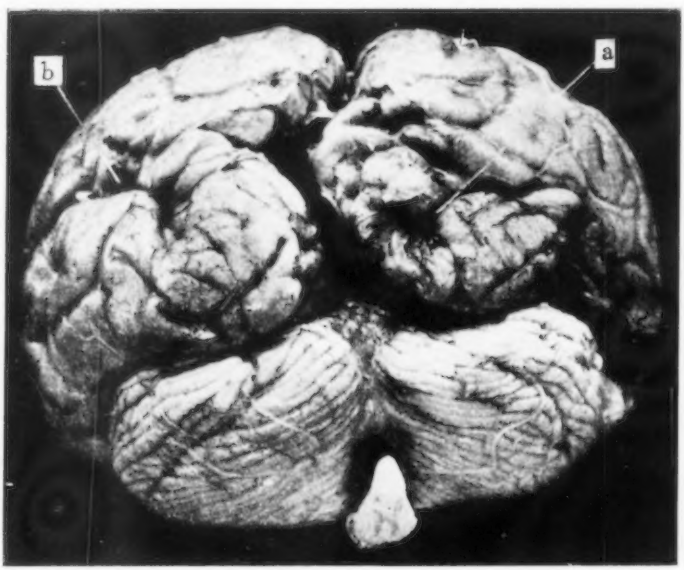

FIG. 5.—Brain in the case of M. M'G., showing the appearance of the occipital lobe as seen from behind. The letter "a" points to the lesion in the right, and the letter "b" to the lesion in the left occipital lobe.

Post-mortem Examination.

This was made on October 22, 1910 (twenty-six years after the occurrence of the cerebral lesion), by Mr. W. Waldie in my presence.

Head.—The dura mater was somewhat thickened and adherent at the vertex.

The arteries at the base of the brain were somewhat atheromatous; a definite patch of atheroma was situated in the right posterior cerebral artery just beyond its junction with the circle of Willis, and another on the left common carotid artery just at its junction with the circle of Willis.

The convolutions of the brain, more particularly of the frontal lobes, were somewhat atrophied and separated by deep sulci—apparently a senile change.

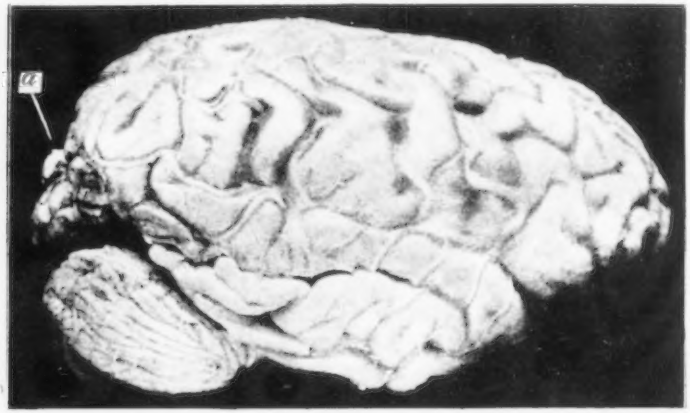

FIG. 6.—Lateral view of the right side of the brain in the case of M. M'G., showing the appearance of the occipital lobe, to which the letter *a* points.

A large lesion was present in each occipital lobe and a small superficial lesion in the adjacent part of the right temporo-sphenoidal lobe (posterior end of the middle temporo-sphenoidal convolution).

In the right occipital lobe an extensive lesion was seen (*see* figs. 5 and 6), a marked depression in the middle of the lobe, and shrinking in of the convolutions, with discoloration, involving more particularly the upper and middle convolutions; the lower convolution at its tip and the visual area (cuneus and calcarine area) appeared to be normal.

In the left occipital lobe there was a marked depression on the upper surface, apparently leading into a cavity in the interior of the brain (*see* figs. 5 and 7). The surrounding convolutions were plump and apparently normal.

Viewed from the side, there was marked depression at the junction of the right occipital lobe with the back part of the temporo-sphenoidal

lobe (*see* fig. 6). A small part of the posterior end of the right middle temporo-sphenoidal convolution was shrunken and atrophied (the result of an old softening).

There was also an area of superficial softening in the middle part of the middle and lower temporo-sphenoidal convolutions on the left side (*see* fig. 7); it was separated from the occipital lobe by an area of normal brain tissue, measuring 1 in. from before backwards.

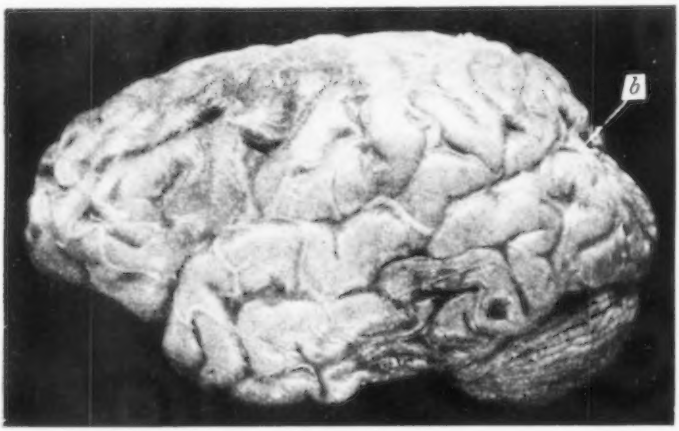

FIG. 7.—Lateral view of the left side of the brain in the case of M. M'G., showing the appearance of the occipital lobe, to which the letter *b* points.

After separating the cerebellum and looking at the back part of the base of the brain from below, there was a marked depression on the right side at the junction of the occipital and temporo-sphenoidal lobes. The convolutions of the occipital lobes (both right and left) on their under surfaces were plump and normal.

The brain was injected with 10 per cent. formalin. On November 12, 1910, it was cut into three sections (*see* fig. 8). The lateral ventricles were greatly dilated. The whole brain was sent to Dr. Shaw Bolton for further examination.

The brain lesion consisted in extensive destruction of the white matter and part of the cortex of the left occipital lobe and very extensive destruction of the white and grey matter of the right occipital lobe. The lesions apparently were the result of old softenings, probably, from the very sudden onset, embolic in origin, with great secondary (compensatory) dilation of the lateral ventricles.

Lungs.—*Right*—the upper lobe, which was adherent, was consolidated by acute croupous pneumonia. On section, the affected area was found to be in a state of grey hepatization, almost gangrenous. *Left*—congested and œdematous, but otherwise normal.

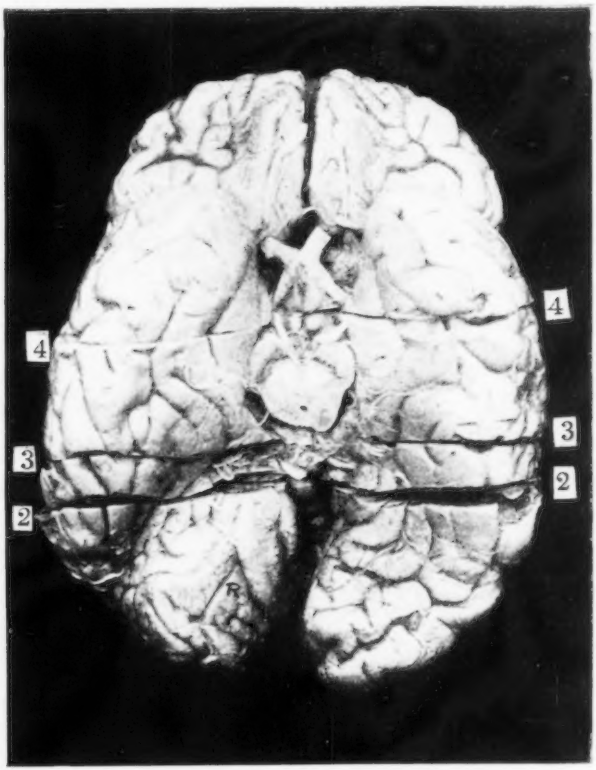

FIG. 8.—Base of cerebrum showing the positions of the transverse vertical sections 2, 2, 3, 3, and 4, 4, the front faces of which are respectively illustrated in figs. 12, 10 and 9. The back face of section 2, 2 is shown in fig. 13. R, cuneus of right hemisphere, which, owing to the lesion, in this position of the cerebrum looks upwards instead of inwards.

Heart.—This showed senile changes but no valvular lesion.

Kidneys.—Both were rather pale and somewhat atrophied, but not markedly abnormal.

Bladder.—This was much thickened and encrusted with phosphates—acute and chronic cystitis.

Histo-pathological Report.

General naked-eye appearances. — The base of the brain shows but one abnormality, namely, a partial rotation of the right occipital pole, which results in the right cuneus (fig. 8, R) looking downwards and inwards instead of inwards. This, as will be seen, is due to the lesion in the right occipital lobe. Sections (front face) through lines

Left Right

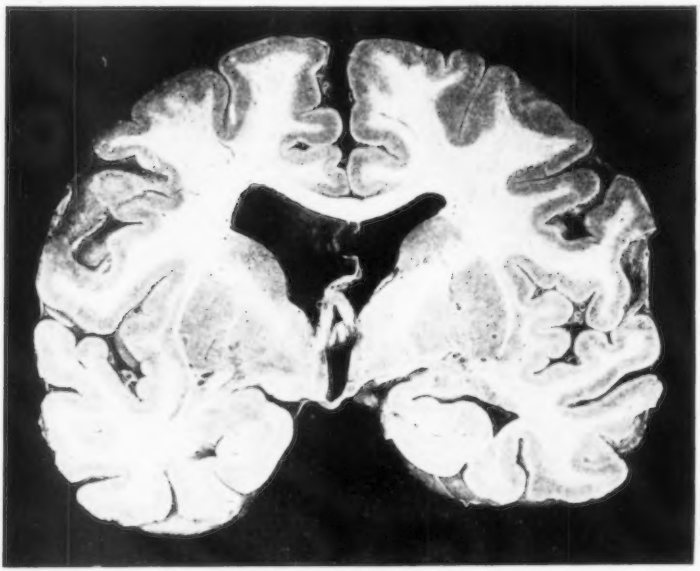

Fig. 9.—Front face of transverse vertical section 4, 4 of the preceding figure. The right lateral ventricle looks smaller than the left both in this figure and in figs. 10 and 12. This is due to the larger size of the lesion on the right side with the greater consequent loss of cerebral tissue and secondary shrinkage which have resulted. No actual lesion can be seen in this figure.

4, 4, and 3, 3, in fig. 8 are shown respectively on figs. 9 and 10. These sections call for no special remark. The lateral ventricles are dilated and there is some degree of lack of symmetry, but even the latter of these figures is anterior to the limits of the lesions.

The optic nerves, basal ganglia, &c., are to the naked eye normal.

The lesions.—These are shown from behind in fig. 11. The left lesion on the stripped specimen is merely a deep fissure or scar on the outer surface of the occipital lobe, and has associated with it a

Left Right

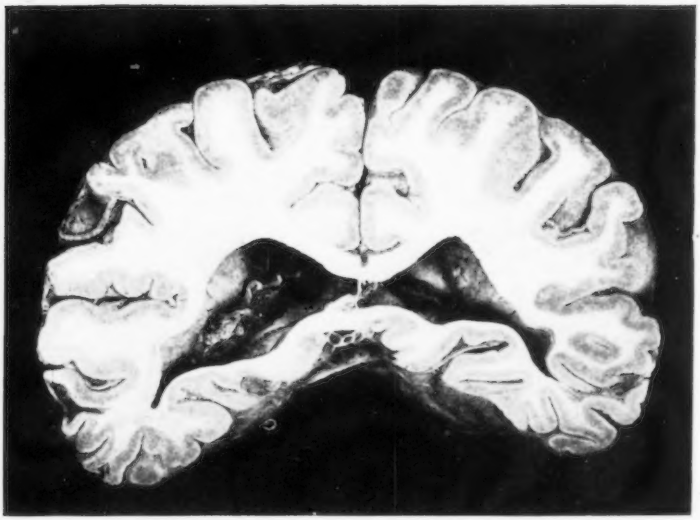

FIG. 10.—Front face of transverse vertical section 3, 3 of fig. 8. The remarks in the legend to the preceding figure apply equally to the present figure.

Left Right

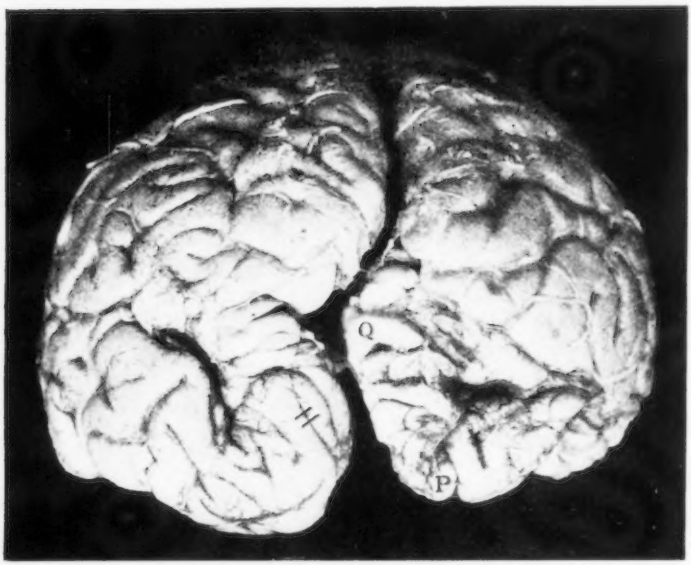

FIG. 11.—Cerebrum from behind, showing the almost vertical left-sided lesion as a deep scar, and the large triradiate scar on the right side. Q, right quadrate lobule; P, pole of right hemisphere; —, upper end of the radiate extremity of the left calcarine fissure. The floor of the extensive right-sided lesion is not visible in this figure. It is shown in figs. 12 and 13 respectively in its natural shrunken and in the stretched state.

folding inwards of the quadrate lobule and the cuneus to an obtuse angle with one another. The right lesion is a much more extensive triradiate scar extending to the pole (fig. 11, P). This scar, in its natural folded-up state, results in a <-shaped projection mesially (fig. 11, Q), of which the upper limb and angle are the quadrate lobule and the lower the cuneus. This projection fits into the depression between the left quadrate lobule and the cuneus. The long posterior extremity of the left calcarine fissure is marked on the figure by a .

Left Right

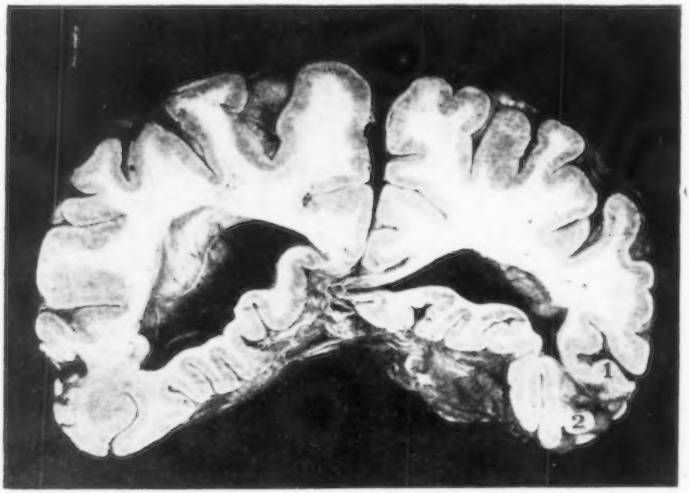

FIG. 12.—Front face of transverse vertical section 2, 2 of fig. 8. This figure shows that the lateral branch of the triradiate lesion of the right side extends forwards to beyond the plane of the section. The numerals 1 and 2 are respectively placed above and below the lesion, the shrunken floor of which is seen between them. As the consequence of this shrinkage the right hemisphere and the right ventricle look smaller than the left hemisphere and the left ventricle. The first of these appearances is real: the second, as the following figure shows, is illusory.

The lateral limb of the triradiate right-sided lesion extends forwards for some distance beyond the line 2, 2, on fig. 8. This is indicated by the numerals 1 and 2 on fig. 12, which represents the anterior face of section 2, 2 on fig. 8, and by the same figures on fig. 13, which shows the posterior face of the section 2, 2 on fig. 8. In the latter figure, the posterior horns of the respective right and left

lateral ventricles are filled with cotton-wool, and thus the extensive floor 1, 2 of the lesion of the right occipital lobe is exposed. The floor of this lesion, when opened out, occupies an area of about 4 sq. in. This lesion, as stated, extends forwards by its lateral limb beyond the line 2, 2 of fig. 8, and by its postero-inferior limb (fig. 11, P) it reaches the pole of the right hemisphere and nearly

FIG. 13.—Back face of transverse vertical section 2, 2 of fig. 8. This figure seen in a looking-glass becomes the duplicate of fig. 12. The ventricles are filled with cotton-wool. The right ventricle is here seen to be the larger, and between numerals 1 and 2 the floor of the lesion, which consists solely of pia-arachnoid and ependyma, is well shown. It is owing to the great loss of tissue caused by the right lesion that the right cuneus looks downwards instead of inwards when the brain stands in its natural position. (fig. 8, R), and that the quadrate lobule projects so markedly inwards (fig. 11, Q).

to the outer extremity of the calcarine fissure of this side. *Nowhere else in either hemisphere* (as will be shown later) *does the lesion come into even the approximate neighbourhood of the visuo-sensory area.*

The visuo-sensory areas and the fissural pattern of the calcarine region of the occipital lobes.—The visuo-sensory areas of the two occipital lobes have been carefully delimited histologically by the method of piecemeal examination, which, though very laborious, is the only really satisfactory one, and a description of these areas will now be given. The necessary account of the fissural pattern of the needed

portions of the respective occipital lobes will be included in this description, as it would serve no useful purpose to give these separately.

As, however, the convolutional patterns of the two sides, as well as the size and shape of the respective visuo-sensory areas, present important differences, it is necessary to deal with these sides quite separately.

Left hemisphere.—In figs. 14, 16 and 18, are respectively shown photographs of the posterior part of the left hemisphere from within, from below and within, and from behind, below and within. Further, in figs. 15, 17 and 19, which exhibit the same views as drawings of two diameters, is shown the exact distribution of the left visuo-sensory area. In all these figures the various numbers are introduced as marks of identification for corresponding regions and fissures. These numbers serve no other purpose and are not the same as those employed in the illustrations of the right hemisphere.

The fissural pattern on the left side is conventional, with the exception of the fact that the posterior cuneo-lingual annectant (situated at 4) is complex and partly superficial, and thereby complicates the posterior extremity of the calcarine fissure.

The visuo-sensory area occupies the strictly normal distribution. It begins anteriorly on the lower lip of the stem of the calcarine fissure, spreading upwards over the apex of the cuneus and downwards towards the collateral fissure (14). Superiorly, it follows the line of the parallel cuneal sulcus (11) (figs 14 and 16), and then that of the commencement (8) and the completion (17) of the superior polar sulcus (fig. 18). It does not, however, occupy the whole of the gyrus enclosed by this sulcus. Here, therefore, the visuo-sensory area is slightly less extensive than usual, or, equally probably, the part of the fissural system 8, iii, 17, where "iii" is placed (fig. 18), represents the sulcus lunatus of Elliott Smith, in which case no marked decrease of extension exists. Inferiorly, the edge of the visuo-sensory area follows the line of the collateral fissure (14) at first, and then, in a quite normal manner, guided by the surrounding sulci, of which 13, 13*a* and 10 (figs. 14 and 16) represent the inferior polar sulcus, it encircles the extremity (marked 4, 7, 6 and 5) of the calcarine fissure (figs. 14 and 16). The fact that the edge of the visuo-sensory area does not reach throughout the gyrus encircled by the inferior polar sulcus indicates a slightly deficient extent of the area, and thus resembles its distribution round the superior limb of the posterior extremity, where it does not occupy the whole gyrus encircled by the superior polar sulcus 8, iii, 17.

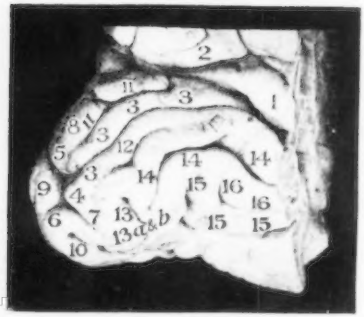

FIG. 14.—Photograph of the left occipital lobe from within. The various numerals are inserted as marks of identification for corresponding regions and fissures in figures 14-19. 1 lies above the stem of the calcarine fissure; and 3 indicates its course, 4 its point of division, and 5, 6 and 7 the portions into which it divides. 5 represents the external portion which appears behind the pole as " " on fig. 11. 11 lies above the parallel cuneal sulcus, and 14 along the line of the collateral fissure.

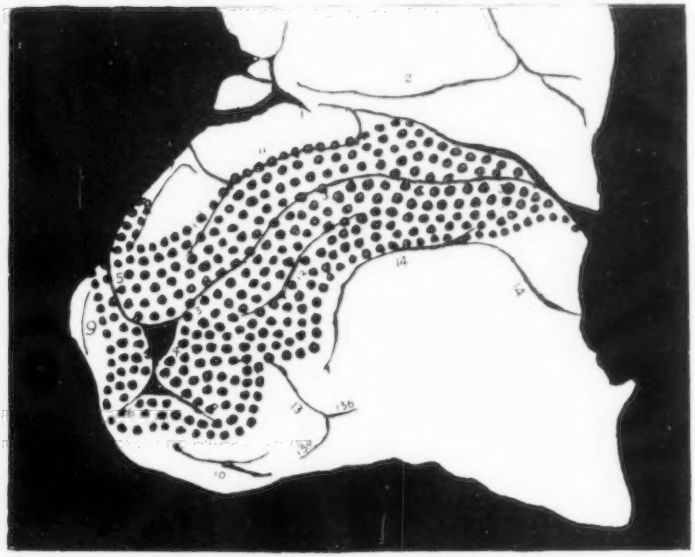

FIG. 15.—Drawing of fig. 14, enlarged two diameters. The numerals correspond with those of the last figure. The visuo-sensory area, in its superficial extension, is indicated by dots. Its normal fissural relationship is well shown. Inferiorly it does not extend as far as the composite inferior polar sulcus 13, 13*a*, 10.

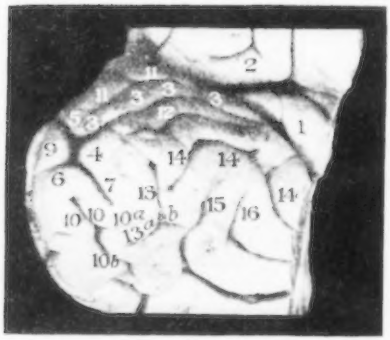

FIG. 16.—Photograph of the left occipital lobe from below and within. The numerals correspond with those in the last figures.

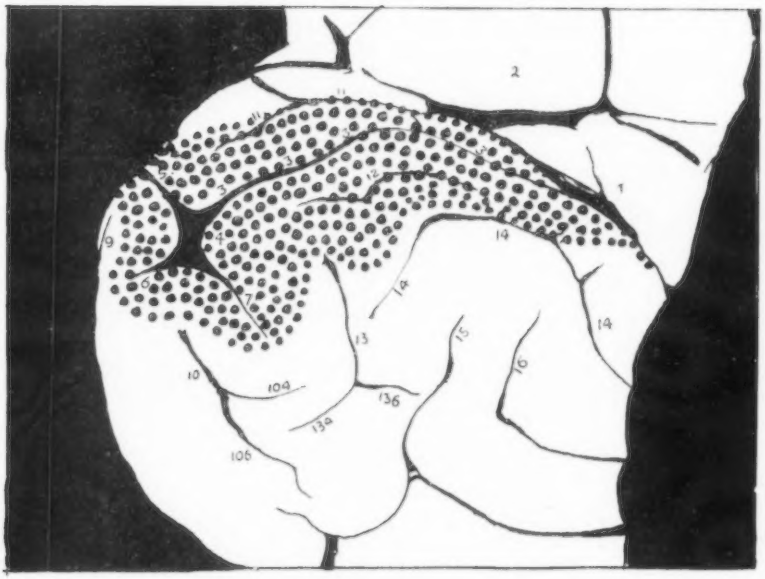

FIG. 17.—Drawing of fig. 16, enlarged two diameters. The visuo-sensory area is better seen in this drawing, and its surface extension in relation to the calcarine stem (below 1), the collateral fissure (11), and the inferior polar sulcus (13, 13*a*, 10*a*, 10), is definitely shown.

Right hemisphere.—In figs. 20, 22 and 24, are respectively shown photographs of the posterior part of the right hemisphere from within, from below and within, and from behind, below and within. Further, in figs. 21, 23 and 25, which exhibit the same views as drawings of two diameters, is shown the exact distribution of the right visuo-sensory area. The various numbers on these figures are introduced as marks of identification solely, as were different numbers in figs. 15, 17 and 19 in the case of the left hemisphere.

The fissural pattern on the right side is less conventional than on the left. The normal truncated appearance so often seen in the right occipital lobe is accentuated, and the calcarine fissure is much shorter than its fellow. In one interesting point, however, a marked resemblance between the two calcarine fissures exists, namely, the complex posterior extremity (4, 12, 11) associated with the large partly superficial and complex posterior cuneo-lingual annectant (fig. 24), which should be compared with the corresponding posterior extremity of the left calcarine fissure (figs. 14, 16 and 18.)

The sulci surrounding the calcarine fissure on the right side are very aberrant. In association with the great width of the cuneus, parallel fissures (14 and 15) exist (figs. 20, 22 and 24), which are at right angles to the site of the usual parallel cuneal sulcus, the only sign of which is apparently the T-shaped mesial extremity of fissure 14. Inferiorly, the collateral fissure (5) is normal in distribution, but fissures 6, 9 and 10, and even 13, are all unusual. Fissure 9 ends quite superficially, however, in the calcarine fissure itself between 12 and 11 (fig. 24). The posterior branch of 13 possibly represents, with 16, a large polar sulcus, but this is uncertain as both these fissures run into the lesion, which, however, just escapes the tip of the calcarine fissure (fig. 24, and also fig. 25).

Allowing for these fissural abnormalities, the distribution and extent of the, however decidedly small, visuo-sensory area of the right side are normal. These, being indicated in the diagrams (figs. 21, 23 and 25), need not be described in detail. It may, however, be remarked that the area extends well forwards along the lower lip of the stem of the calcarine fissure in an absolutely normal manner (fig. 23).

Histo-pathology.

Before dealing with the visual cortex, it may here be remarked that the infra-cortical visual apparatus is normal.

The *optic nerves* contain abundant nerve-fibres throughout. Com-

Lesion

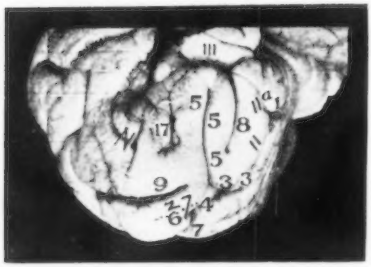

FIG. 18.—Photograph of the left occipital lobe from behind, below and within. The numerals correspond with those of the preceding figures. The letter N is placed just below the inferior extremity of the lesion on this side.

Lesion

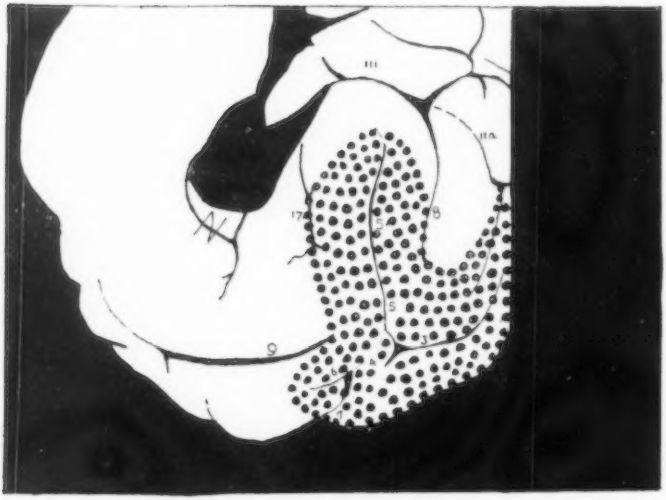

FIG. 19.—Drawing of fig. 18, enlarged two diameters. The posterior extremity of the visuo-sensory area, especially that behind the region of the posterior cuneo-lingual annectant (situated at 4) is clearly defined, as is the relationship of the top of the area to the well-defined superior polar sulcus (17, 111, 8). The large black area above the letter N represents the lesion on this side, and the figure thus clearly indicates that the visuo-sensory area is intact in its entirety.

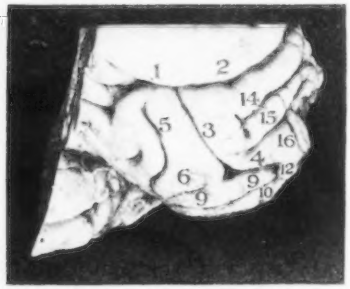

FIG. 20.—Photograph of the right occipital lobe from within. The various numerals in figs. 20 to 25 indicate corresponding points in these figures and thus serve a similar purpose to the numerals employed in the previous set of figures. 1 lies above the calcarine stem, and 2 above the parieto-occipital fissure. 3, 4, 12 indicate the course of the calcarine fissure. 5 indicates the collateral fissure, and the inner ends of 14, 15 the aberrant caneal sulcus.

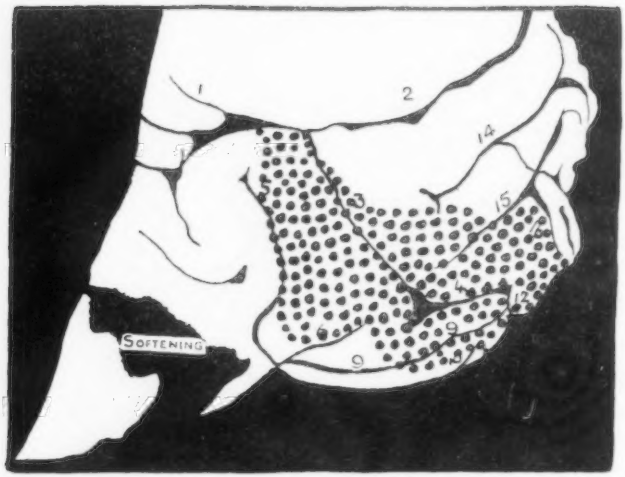

FIG. 21.—Drawing of fig. 20, enlarged two diameters. The numerals correspond with those of the last figure. The visuo-sensory area, in its superficial extension, is indicated by dots. The word "softening" is placed over a local lesion in the left of the figure. The fissuration of this side is rather aberrant, and it should be noted that a similar remark applies to the distribution of the visuosensory area, though to a much less extent.

pared, however, with normals they show some irregularity in shape and a considerable decrease in the size and number of the normal subdivisions of fibrous tissue, together with much fibrosis of the vessels.

The utmost that can be said, from the aspect of morbid histology, is that they exhibit what would be expected after prolonged disuse in the absence of neuronic lesions.

The optic radiations are normal, and the cortical lesions nowhere approach these.

The cortex[1] of the *visuo-sensory* area exhibits striking characteristics. That quite adjacent to and within the calcarine fissure is, as far as can be determined histologically, normal. The whole of the peripheral portion is much thinned, in a more or less patchy manner, but the general characteristics are those of a simple disuse atrophy involving the outer cell or pyramidal lamina (L. II) especially, but to a considerable extent also the outer granule layer (L. IIIa) and the outer fibre or superficial lamina (L. I).

This appearance extends throughout the area, with the result that in general terms the pear-shaped visuo-sensory area consists roughly of a calcarine core of practically normal cortex and of a more or less atrophied surface extension.

This general description applies to both sides of the brain and, as far as can be ascertained, to an equal degree in both.

On the right side, however (see figs. 21, 23 and 25), the area is smaller than on the left (figs. 15, 17 and 19), and its posterior extremity (though not the calcarine fissure) is involved in the lesion (fig. 25).

The surrounding visuo-psychic cortex exhibits no abnormality beyond a slight and variably distributed degree of disuse atrophy of L. II, the outer cell or pyramidal lamina.

Concluding Section.

The fact that the visuo-sensory area generally is normal in distribution makes it certain that the gross contraction of the visual fields which is found clinically is due to an associative block, and that this block is not quite so complete on the left side of the visual apparatus as on

[1] The cortex of the visuo-sensory area consists of the following layers:—
L. I. *Outer fibre lamina*, or superficial layer.
L. II. *Outer cell lamina*, or pyramidal layer.
L. III. *Middle cell lamina*, consisting of the following layers: L. IIIa. Outer layer of granules. L. IIIb. Line of Gennari or "optic" fibre layer. L. IIIc. Inner layer of granules.
L. IV. *Inner fibre lamina*, or inner line of Baillarger.
L. V. *Inner cell lamina*, or polymorphic layer of nerve-cells.

FIG. 22.—Photograph of the right occipital lobe from below and within. The numerals correspond with those of the last two figures.

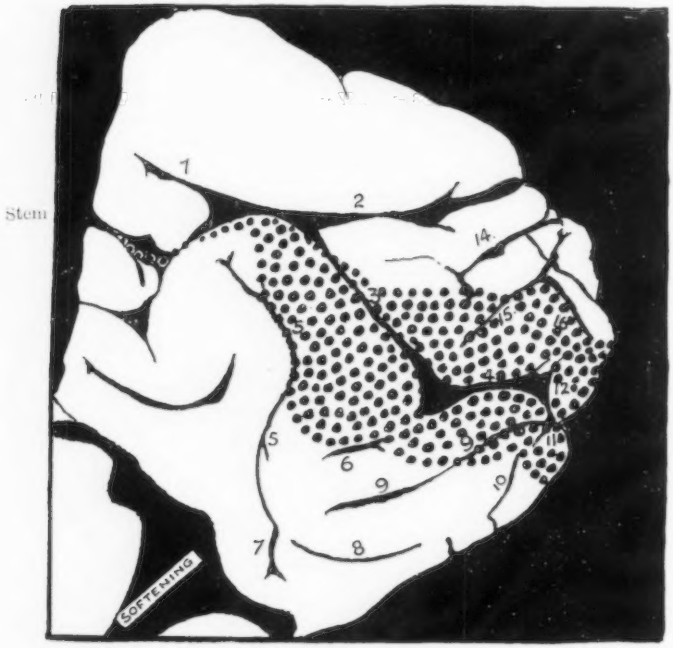

FIG. 23.—Drawing of fig. 22, enlarged two diameters. The general distribution of the visuo-sensory area is better seen in this figure than in fig. 21, and it should be noted that the area extends forwards, as is shown by the white rings, almost to the line of the cross-section at the point where the word "stem" is placed. It is thus incidentally evident, as the fissuration shows, that 1 is in this figure placed not above the calcarine stem, but above a small sub-fissure considerably above it. The word "softening" is again placed over a local lesion distinct from the main softening.

FIG. 24.—Photograph of the right occipital lobe from behind, below and within. The numerals correspond with those of the last figures.

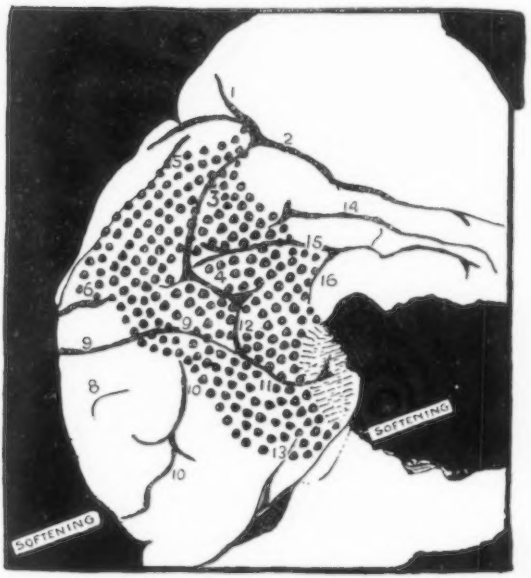

FIG. 25.—Drawing of fig. 24, enlarged two diameters. The word "softening" on the left refers to the small lesion shown in figs. 23 and 21. The same word on the right indicates the posterior limits of the important triradiate lesion already illustrated in general view in fig. 11. The hinder part of the visuo-sensory area is well shown in this figure (line 3, 4, 12, 11). Beyond 11, just at the tip of the calcarine fissure, the first indications of softening begin, but the visuo-sensory area is histologically recognizable right up to the edge of the black area of softening.

the right (and in the right fields of vision as in the left), in association with a smaller or less important lesion on the left side of the cortex. This is indicated histologically by the disuse atrophy of the visuo-sensory cortex which is everywhere marked, with the exception of the more central (calcarine) portion.

It would therefore appear that the calcarine core of the visuo-sensory area serves in some degree as an anatomical basis for macular as distinct from non-macular or panoramic vision.

This conclusion is supported by the fact that the lesion in the right hemisphere reaches up to the posterior portion of this calcarine core. This fact at the same time incidentally explains why in the case the right field of (central) vision was a little more extensive than the left.

It appears, therefore, to be probable that human macular vision is an evolution dependent on (1) the development of binocular vision, i.e., on the employment of corresponding parts of the retinæ simultaneously; and on (2) the simultaneous development of the capability to pay prolonged and individual attention to particular points of the general visual panorama.

Macular vision is thus superposed on the neuronic apparatus for panoramic, and the two types shade into one another, the central parts of the retinæ and the corresponding central (calcarine) cores of the pear-shaped visuo-sensory areas being the parts concurrently employed in macular vision.

The associative block caused by the lesions in this case, therefore, being greater on the right side than on the left, must thus be regarded as having been enough to stop the passage of the feebler panoramic stimuli without interfering seriously with the macular and stronger visual stimuli.

The continued passage of the latter has prevented the onset, in the calcarine core of the visuo-sensory area, of the disuse atrophy which elsewhere in these areas is general.

Lastly, the slight maiming of the right calcarine core posteriorly is responsible for the fact that the clinical visual field of the case was rather less extended on the left side.

The histological investigation of this case may thus claim not only to have explained the clinical features present during life, but to have added to our knowledge of the part played by the visuo-sensory area in macular and in non-macular or panoramic vision. It may, in fact, be stated that the anatomical basis of the former is the cortex of the calcarine core of the pear-shaped visuo-sensory area, and that the anatomical basis of the latter lies in the surrounding and remaining visuo-sensory cortex.

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